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THE MANAGEMENT OF FACIAL PARALYSIS ASSOCIATED WITH FRACTURES OF THE TEMPORAL BONE.*

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Definitive surgical treatment of facial paralysis due to basilar skull fracture is necessary in certain instances; and in these few properly selected cases, it may be accomplished with gratifying results.

Facial paralysis associated with acute head trauma indicates temporal bone fracture in nearly every instance. It is usually accompanied by bleeding from the ear on the involved side and rather rarely by cerebrospinal otorrhea.

The incidence of facial paralysis produced by acute head injury is somewhat difficult to determine, but a review of reported series of cases would indicate that it is relatively high if one considers only the middle and posterior fossa fractures. Grove[1] reported 29 cases of facial paralysis in his series of 211 skull fractures (14 per cent). In his exhaustive review of the literature pertaining to fractures of the petrous bone, he cited the statistical studies of many authors. He said: "According to the statistics of Siebenmann, Brunner, Voss, and

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Ulrich, transverse fractures cause a facial paralysis in about 50 per cent of the cases, and in the longitudinal variety in from 10 per cent to 18 per cent." A much higher incidence was recorded by Davis[2] who reported peripheral facial paralysis in 46 per cent of middle fossa fractures.

Even in large series of unselected cases of acute head injury, the incidence of facial paralysis is considerable. Turner[3] reported 46 instances in his series of 1550 acute head injuries of all types. With the exception of the olfactory nerve which was injured in 119 instances, it was the cranial nerve most commonly involved. In a series of 600 cases of head injury, Russell[4] found that facial paralysis occurred 17 times. It was interesting to note that this represented about one-fifth of the cases in which there had been bleeding from the external ear.

The most common type of skull fracture which may involve the facial nerve is one in the floor of the middle fossa. Rowbotham[5] states that in his experience, these are more apt to extend from the vault to the base than to be confined to the base alone. They may start in the temporal squama or the parietal bone and extend through the mastoid into the middle ear. Rowbotham states that the facial nerve is usually injured distal to the geniculate ganglion. Ulrich[6] stated that when the facial nerve is injured, the injury is in the region of the geniculate ganglion. Because of the strong buttress effect of the petrous pyramids, fracture lines extending from the vault are apt to be deflected horizontally along the anterior border of the petrous. Such horizontal fractures occur much more frequently than transverse fractures; thus the middle ear is involved much more commonly than is the otic capsule. The transverse fractures are primarily posterior fossa fractures which have resulted from terrific force applied through the condyles.

Kettel[7] describes two types of transverse fractures: the internal which involves the internal auditory canal and the cochlea; and the external which passes through the cochlea, vestibule, and Fallopian canal. In these, there is no tearing of the tympanic membrane and hence no bleeding from the external auditory canal, although there may be a hemotympanum.

Gurdjian[8] describes cases of combined transverse and longitudinal fractures that have come under his observation. In such fractures, there may be wide variations in the neurological signs, depending upon the position and extent of the fracture lines.

Ramadier and Caussé[9] describe an unusual type of fracture confined to the mastoid process which could involve the external canal and middle ear, injuring the facial nerve in its vertical course.

Facial paralysis complicating fractures of the temporal bone may be immediate or delayed. The immediate paralyses obviously denote a direct injury at the time of fracture which may represent laceration, stretching, or compression of the nerve by bone fragments. The delayed cases may be due to edema and hemorrhage, or, still later and less commonly, to infection.

There is an abundant literature on all aspects of skull fractures. Even a cursory review of articles dealing with fractures of the temporal bones is beyond the scope of this presentation. For the present purpose it has seemed sufficient to cite authoritative descriptions of the most common types of temporal bone fractures, and to note accepted opinions regarding the frequency of associated paralysis and the most common sites of injury to the nerve.

More important, perhaps, in consideration of the cases to be presented at this time, is a brief discussion of the prognosis of facial paralysis produced by a fracture through the temporal bone.

Kahn[10] and Gurdjian[8] agree that about 90 per cent of these cases recover completely, or at least quite adequately. Perhaps three or four of the remaining 10 per cent suffer complete and permanent paralysis, while the rest have poor or inadequate return of function. Kettel,[7] after an exhaustive study of the subject, concluded that of the immediate paralyses, 75 per cent of patients recovered completely; 15 per cent showed partial recovery, and in 10 per cent massive paralysis remained.

In Grove's[1] series of cases, there were 29 instances of facial

paralysis of which five were due to transverse fractures, or at least fractures which extended through the labyrinth, and 21 due to longitudinal fractures. In one transverse and in five longitudinal fractures, the paralysis was complete and permanent. The others "either improved or cleared up completely."

Rowbotham[5] states that at least 90 per cent of cases recover. In his experience, only one case in 30 failed to recover, and that was in an individual who had a fracture through the internal auditory canal. He states that the "favorable cases show signs of improvement within two months and usually within two or three weeks." Rowbotham[5] declares further that "an incomplete injury of the facial nerve may later lead to facial spasm, a condition equally as disabling, and probably more disfiguring than facial paralysis." He concludes that although theoretically, routine decompression should be done in all cases, particularly the delayed ones, early operative interference is contraindicatd because of the high percentage of good spontaneous recoveries; however, he does advocate exploration of the nerve regardless of electrical reactions, after three months, in those patients who have shown no improvement.

Russell[4] expects spontaneous recovery of all forms of facial paralysis after injury, and does not mention decompression or other surgical procedures to alleviate the condition in his chapter in Brock's text.

Spurling[11] states that the prognosis for complete recovery of immediate cranial nerve palsies, due to skull fracture, is poor.

Browder[12] states that "except in rare instances when facial paralysis results from direct trauma to the facial nerve, surgical intervention is contraindicated. Satisfactory recovery may be expected to follow massage and electrical stimulation of the paralyzed muscles."

Turner[13] studied 70 consecutive cases of traumatic facial palsy of which 36 were immediate and 34 were delayed from two to eight days after injury. Of the latter group, 22 were partial and 11 were complete. In the delayed cases one patient,

who developed an acute otitis media on the fifth day, failed to recover any function of the facial muscles, and two had incomplete recovery with mass motion after six and eight months. Of the 36 immediate cases, 19 complete and 17 partial, six had incomplete recovery which started at three months, and three remained completely paralyzed.

If the prognosis of facial paralysis due to skull fracture is good for spontaneous recovery in 90 per cent of cases, and this seems rather high after reviewing the literature on the subject, there is still that 10 per cent of such patients in whom surgical intervention is indicated when the lesion is surgically accessible. By careful assessment of the individual cases, the otologist should be able to select with discrimination those patients in whom exploration of the Fallopian canal is required.

The selection of patients for such a procedure demands careful attention to the details of history and examination. One must ascertain whether or not there was bleeding from the external ear at the time of the accident and if the paralysis was immediate or delayed, for nearly all patients who experience a delayed paralysis proceed to a satisfactory spontaneous recovery. Since specific information regarding the time of onset of the paralysis is too frequently omitted in the history of referred patients, it is felt that, generally speaking, a three months' waiting period is advisable before consideration of surgical intervention. Any physiologic block or neuropraxia should have recovered by that time. In most reported series of cases complete or satisfactory spontaneous recovery, if it is going to occur, is manifest in four to eight weeks. If, however, a response to Faradic stimulation which was previously absent has become demonstrable at the end of three months, further delay may be indicated but is probably inadvisable, for in such patients the degree of spontaneous return of function is likely to be unsatisfactory. The waiting period of three months does not militate against a good result from decompression, nerve suture, or nerve graft if such should be found necessary.

Examination should include careful otoscopic observation, Roentgenograms including Stenver's views, tuning fork and audiometric hearing tests, vestibular tests, percutaneous Far-

adic stimulation, and tests for lacrimation, taste, and salivation. In late cases, muscle responses to galvanic stimulation must be determined also.

Transverse fractures through the labyrinth resulting in facial paralysis with complete loss of cochlear and vestibular function usually carry a bad prognosis, for the site of nerve injury is likely to be inaccessible for repair in continuity. In these instances, substitution anastomoses with the hypoglossal or spinal accessory nerves may be required. Occasionally, however, repair in continuity in such a case may be possible if the transverse fracture is of the external type as described by Kettel.[7] An example of this type is described in Case 3.

Roentgenograms will usually demonstrate well a transverse fracture, but a longitudinal one may be more difficult to detect. Oblique films of the temporal bones are most apt to show those fracture lines extending from the vault into the middle fossa.

If cochlear and vestibular functions are preserved in a patient with facial paralysis due to temporal bone fracture, the facial nerve lesion is probably in a surgically accessible region.

The most valuable single test of facial nerve function has been Faradic stimulation. If good response to Faradism can be obtained, even if the use of 40 volts is required, the prognosis for at least some degree of spontaneous recovery is good. If the Faradic response is absent, it does not indicate the severity of the lesion. I agree with Kettel[7] who states that "a positive response may be a good prognostic sign, but nothing final can be deduced from a negative reaction." Electromyographic studies are valuable, but have not proved to be infallible. They have been of value during the period of waiting, for responses may be obtained prior to those induced by Faradism.

When, after three months, or better at an earlier date if the patient's general condition is good, if the paralysis has remained complete and there is complete reaction of degeneration, surgical exploration must be considered. At this time the localization of the lesion must be determined as accurately as

possible. Tests for chorda tympani and stapedius nerve functions are valuable. For the stapedial nerve test, the binaural stethoscope is used as described by Tschiassny.[14]

Since taste testing for chorda tympani function has been unsatisfactory in many instances, we have been using in addition a technique for measuring the salivary secretion of the submaxillary glands. In this procedure, a No. 60 polyethelene catheter is placed in Wharton's duct on each side. The patient is asked to suck on a lemon for five minutes, during which time the output of saliva is measured. In cases of absent chorda tympani function we have found, as would be expected, a marked decrease in the amount of saliva secreted on the involved side. One reason which may account for some unreliability of testing taste on the anterior two-thirds of the tongue, to determine the function of the chorda tympani nerve, is explained by Crosby.[15] She states that in embryological development some fibers of the glossopharyngeal nerve may be drawn forward in the tongue and serve to conduct gustatory impulses in the absence of chorda tympani innervation.

When surgical exploration with anticipated nerve repair by decompression, end-to-end suture, or grafting is contemplated, the most important determination will be in regard to the function of the lacrimal gland. The Schermer test is very satisfactory, but much information can be gained from observation of lacrimation after irritating the nasal mucous membrane, either chemically or by means of a cotton tipped applicator. If there is a dry eye, and particularly if this is associated with loss of cochlear and vestibular function, exploration will probably be fruitless and nerve repair impossible, except by means of substitution anastomosis. Normal tearing indicates a lesion distal to the geniculate ganglion which should be accessible and amenable to surgical repair in continuity.

Usually a radical mastoidectomy is required, but in some instances simple mastoidectomy may permit of adequate exposure (see Case 1). This, of course, may be anticipated if it can be shown that the lesion is distal to the branch of the facial nerve supplying the stapedius muscle. In some cases, a modi-

fied radical mastoidectomy will be required. Every effort should be made to preserve the patient's residual hearing.

After such careful evaluation, one can proceed with some confidence in those patients in whom surgical intervention is deemed advisable.

Examples of facial paralysis demonstrating different types of nerve injury produced by temporal bone fractures will be presented in three case reports. The surgical technique used in each case was different from the others, and depended upon the pathological condition present.

Case 1: D. LeV., No. 811177, a white male, aged 22, was admitted to University Hospital on May 18, 1955, because of a complete right peripheral facial paralysis. On February 24, 1955, he had sustained a severe head injury with a linear fracture in the right temporal region from an automobile accident. It was reported that the facial paralysis had been immediate and that there had been bleeding from his ear at the time.

Routine objective examination of the pharynx, nose, and left ear revealed no significant pathological changes. The right tympanic membrane was intact although somewhat retracted, and the external auditory canal appeared to be normal. An audiogram showed hearing that was slightly depressed but nearly normal. The vestibular function was normal on caloric stimulation. He had a complete right peripheral type of facial paralysis with complete reaction of degeneration. No response was obtained on percutaneous stimulation with Faradic current of 40 volts. Other than the facial paralysis there was no neurological deficit. The patient had retained sense of taste on the anterior two-thirds of his tongue.

Roentgenograms revealed a fracture line in the right temporal region, which extended downward into the mastoid. The mastoid process appeared to be normally aerated (see Fig. 1).

Because of the continued complete peripheral facial paralysis which apparently had its onset at the time of the injury, the complete reaction of degeneration and absence of injury to the otic capsule, indicating that the traumatic lesion to the facial nerve was accessible, surgical exploration with an effort to effect nerve repair was advised.

On May 20, 1955, approximately three months after injury, a right mastoidectomy was performed. There was no evidence of any infection in the large pneumatic mastoid process. The fracture line was clearly visible as it extended over the mastoid cortex to the upper portion of the external auditory canal. In enlarging the aditus ad antrum, it was noted that the fracture had extended into the fossa incudis. After removal of the mastoid tip, the facial nerve was exposed throughout its vertical course and appeared to be normal up to the pyramidal segment. Further exploration in the region of the fossa incudis revealed that the short crus of the incus had been dislodged and that there were small spicules of bone in the region of the fossa, which were compressing the facial nerve. After removing these spicules the exposed nerve in this region appeared to be compressed to about one-third its normal size (see Fig. 2).

A little further rotation of the incus without disarticulating it, per-

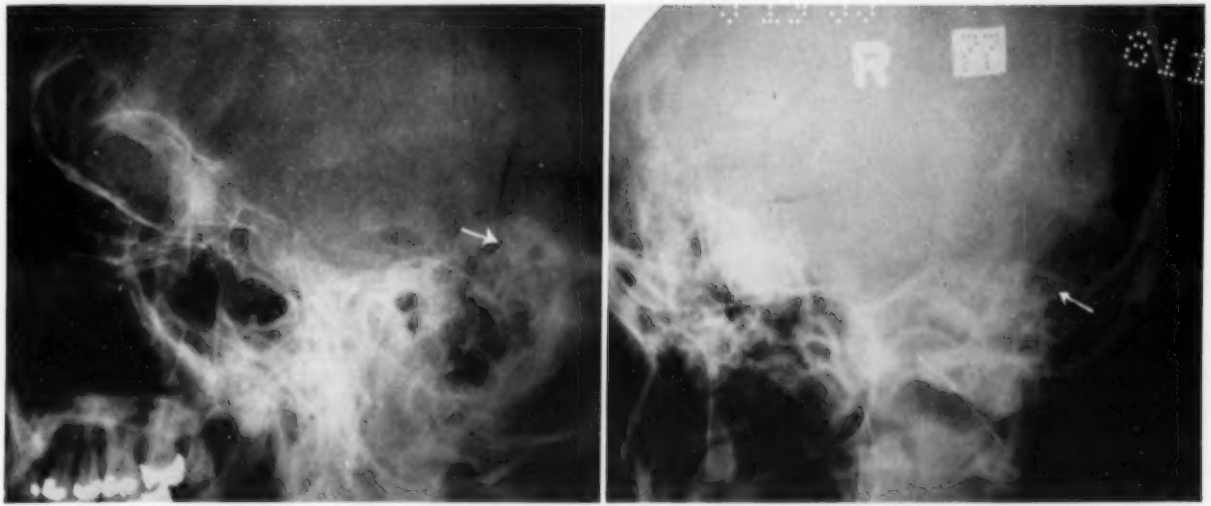

Fig. 1. Case 1. Roentgenograms demonstrating the fracture line in the temporal squama extending into the mastoid process indicated by arrows.

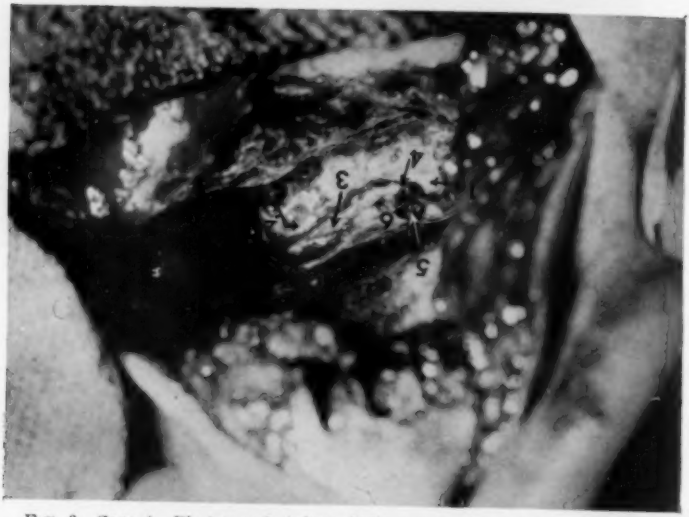

Fig. 2. Case 1. Photograph taken during operation after completion of mastoidectomy and exposure of facial nerve.

Arrows indicate: 1. Horizontal semicircular canal; 2. Mastoid tip; 3. Vertical course of facial nerve; 4. Compressed nerve at pyramidal segment; 5. Incus; 6. Elevated short crux of incus.

Fig. 3. Case 1. Photograph taken at end of operation showing bulging of facial nerve after slitting the sheath.

Arrows indicate: 1. Horizontal semicircular canal; 2. Mastoid tip; 3. Vertical course of facial nerve with sheath opened; 4. Bulging pyramidal segment of facial nerve; 5. Incus; 6. Middle ear cavity.

mitted sufficient exposure to uncap the facial nerve in its entire intratympanic course and to examine the ossicular chain, which was found to be intact. The nerve had an essentially normal appearance, except in its pyramidal segment. Under magnification, the sheath of the nerve was slit from the stylomastoid foramen upward throughout its intra-

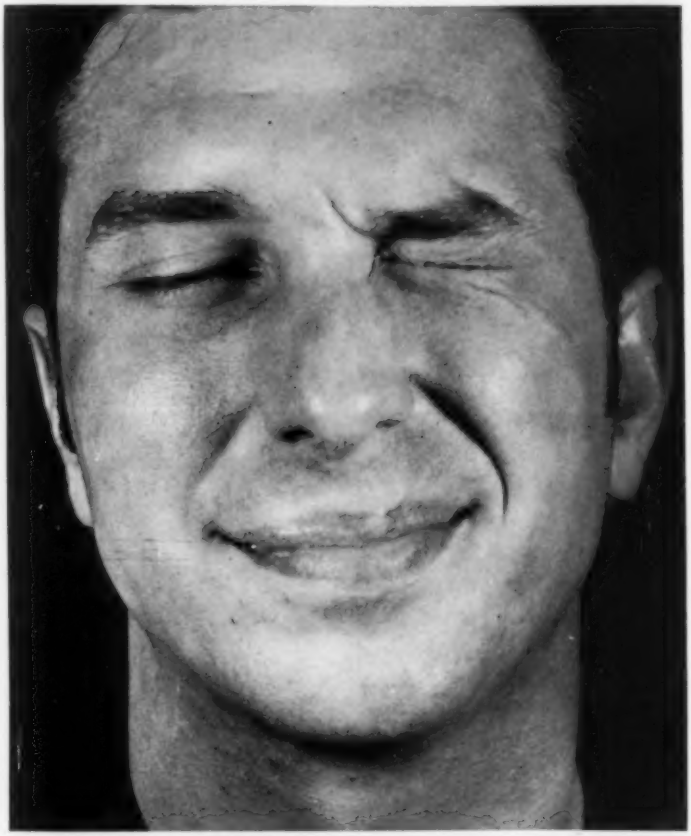

Fig. 4. Case 1. Photograph of patient one month after operation.

tympanic course. After this neurolysis the nerve bulged out to its full normal-appearing size in the region where it had been compressed (see Fig. 3). No evidence of gross damage to the nerve fascicles was apparent.

The patient's convalescence was uneventful. He was discharged from the hospital on the fifth postoperative day, at which time a little flicker of motion was noted in his upper lip.

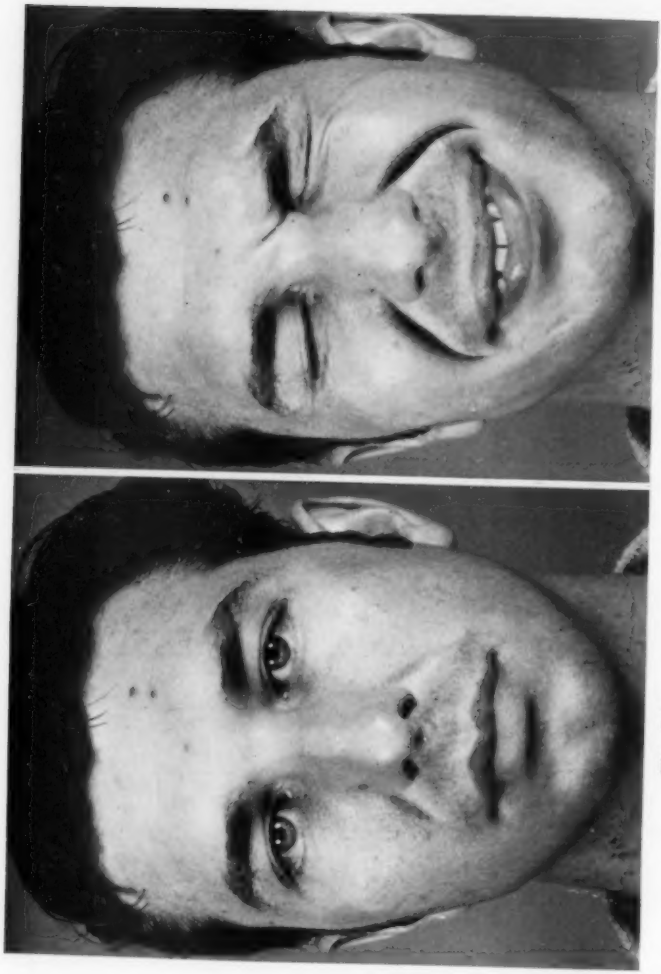

Fig. 5 A. Fig. 5 B.

Fig. 5. Case 1. Photograph of patient taken 3 months after operation showing further improvement.

By June 22, 1955 (one month postoperative) the patient showed considerable return of function. He could close his eye tightly but did not have as much power on the operated side of the face as he did on the normal side (see Fig. 4). There had been preservation of the preoperative hearing.

He was last examined on August 8, 1955, at which time his general condition was excellent. Functional return of facial muscles was adequate but not complete, in that there was weakness of the lower lip, weak frontalis action, and a little mass motion (see Fig. 5). The audiogram showed nearly normal hearing, as seen in Fig. 6.

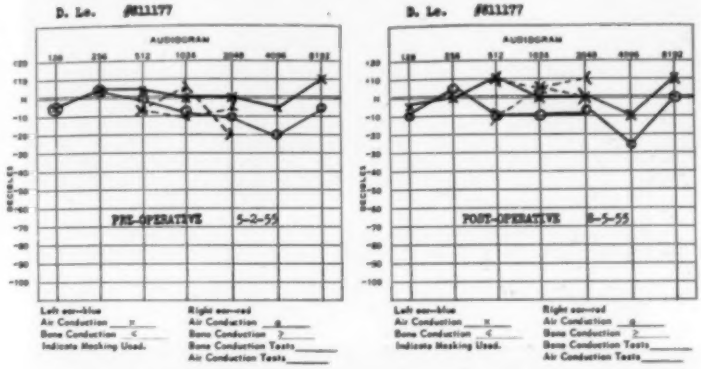

Fig. 6. Case 1. Pre operative adiogram, 5-2-55. Post-operative audiogram, 8-5-55.

Case 2: R. W., No. 736200, a white male, aged 14, was admitted to the University of Michigan Hospital on Sept. 20, 1952, with an acute head injury. Earlier the same day a jack had slipped, allowing the car on which he was working to fall on his head. Examination revealed a partial left peripheral type of VIIth nerve palsy, paralysis of the left Vth and VIth cranial nerves and of the right second and third cranial nerves. Radiographs of the skull demonstrated multiple fractures on the right side, and a fracture on the left side extending from the left temporoparietal area into the mastoid (see Fig. 7).

During the next four days the facial paralysis became complete. Otoscopic examination revealed a deformity of the left external auditory canal in its supra-posterior aspect. The left tympanic membrane was dull but intact. Tuning fork and audiometric tests demonstrated a mixed type of deafness on the left side with an air threshold averaging 53 db loss for the speech frequencies.

On Oct. 30, 1952, the facial paralysis was still complete, and there was complete reaction of degeneration (see Fig. 8). Electromyography indicated complete denervation of the left facial muscles. Testing with Faradic current up to 40 volts failed to elicit any facial response. Decompression of the facial nerve was advised.

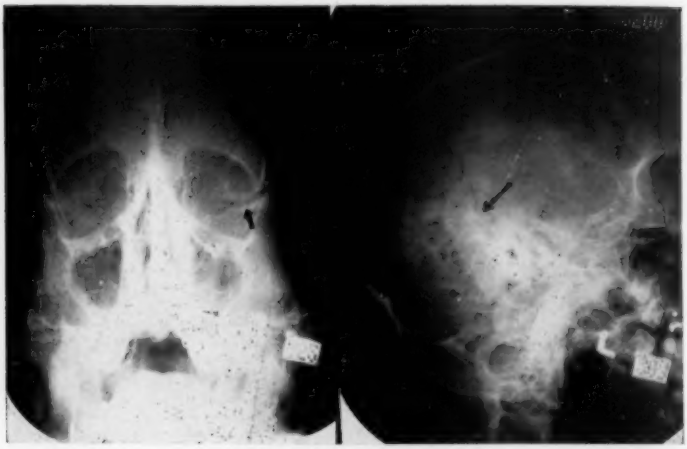

Fig. 7. Case 2. Roentgenograms showing: Fracture line extending from left temporoparietal area into the mastoid. Fracture involving right orbit.

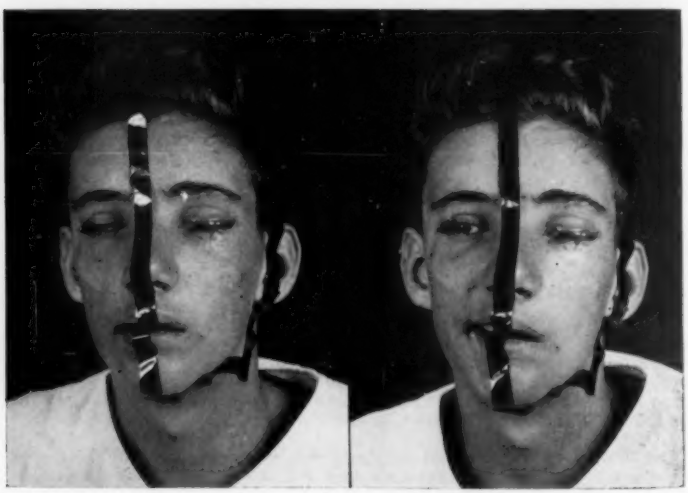

Fig. 8. Case 2. Pre-operative photograph demonstrating left facial paralysis.

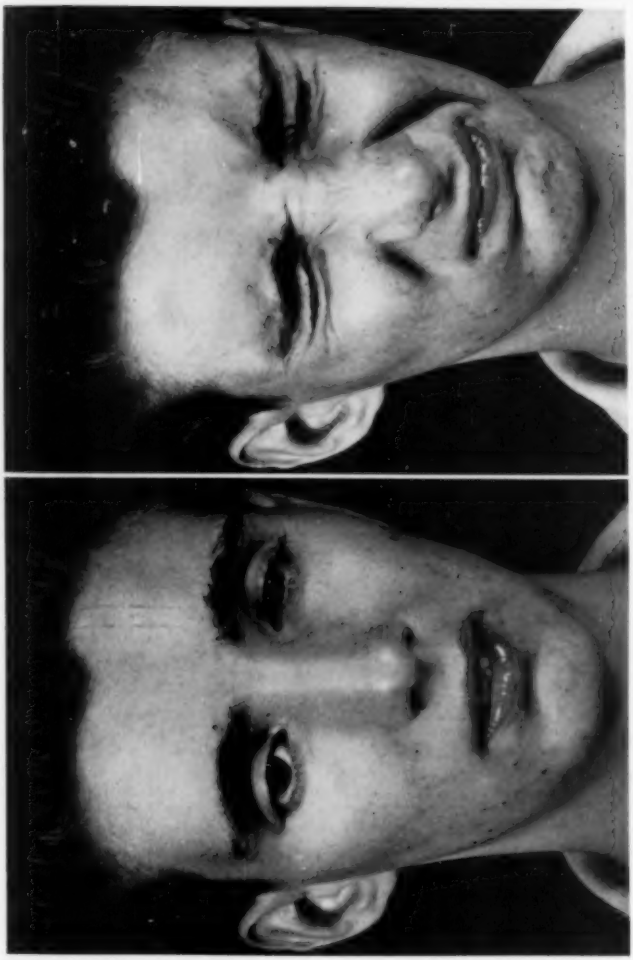

Fig. 9. Case 2. Post-operative photograph, 9-2-55. Note that tarsorraphy on left is still intact.

At operation, performed on Oct. 31, 1952, a fracture in the left temporal squama was found extending into the mastoid bone and through the posterior superior aspect of the external auditory canal. The fracture also extended through the pyramidal segment of the Fallopian canal, then through the tegmen of the middle ear lateral to the geniculate ganglion, and then forward toward the petrous apex. The incus, which had been dislocated anteriorly by the injury was removed. After the completion of a modified radical mastoidectomy, decompression of the facial nerve was accomplished by slitting its sheath. The nerve appeared to be normal from the stylo-mastoid foramen up to the pyramidal segment, but in the intratympanic portion it was grossly edematous and bulged through the opened sheath.

The patient had an uneventful convalescence and was discharged from the hospital on the eleventh postoperative day. Four weeks after operation facial movements were first noted and gradually improved to a maximum during the next four weeks. His most recent examination on Sept.

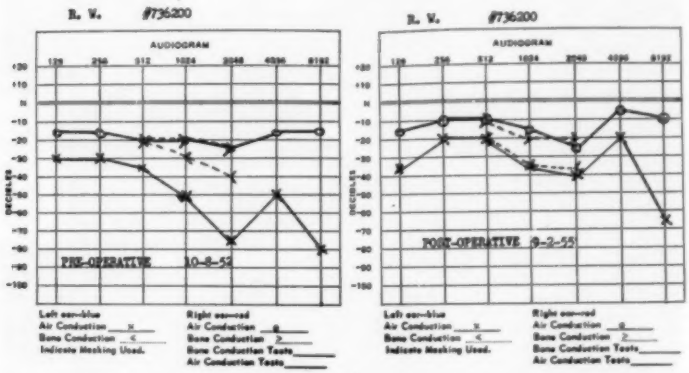

Fig. 10. Case 2. Pre-operative and post-operative audiograms.

2, 1955, indicated good symmetry of the face in repose. He was able to close both eyes tightly and to show his teeth, purse his lips, and wrinkle the forehead on one side as well as on the other (see Fig. 9). There was minimal mass motion and a slight spasmodic tic of the upper and lower lip on the operated side. An audiogram on this date showed an air threshold of 31 db average loss for the speech frequencies in the left ear (see Fig. 10). The mastoid cavity was healed and dry.

Case 3: L. M., No. 811204, a white male, aged 34, entered the University of Michigan Hospital on May 30, 1955, because of a left facial paralysis. He had been well until Dec. 17, 1954, when he fell about 12 feet from the top of a boiler on which he was working. He struck a scaffold, fracturing his left shoulder and then struck the left side of his head on the floor, which caused a skull fracture. It was reported that he was unconscious for about five days and that he bled from the nose, the left eye, and the left ear. There was no history of cerebro-spinal otorrhea. The patient was told that the paralysis of the left side of the face was im-

mediate. Since the accident, the patient had been deaf in the left ear, but had not noted any dizziness.

Routine examination of the pharynx, nose, and right ear revealed no significant pathological changes. There was no paralysis of the tongue,

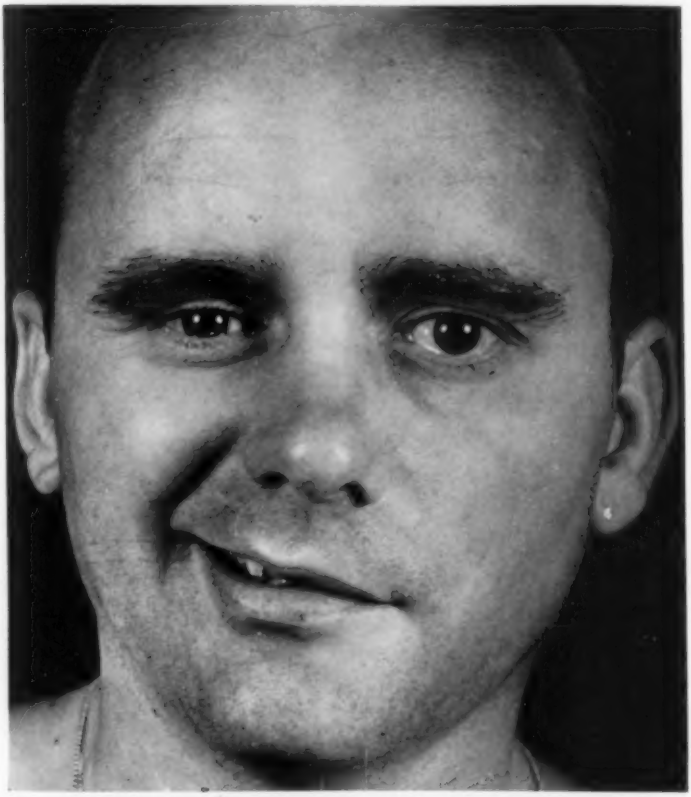

Fig. 11. Case 3. Pre-operative photograph showing left facial paralysis and obvious tearing of left eye.

palate, or larynx. There was a deformity of the left external auditory canal, appearing as an angular irregularity in its posterior aspect at the annulus, where there was a little granulation tissue, but no gross purulent exudate. The patient had a complete peripheral left facial paralysis and no response of the facial musculature could be elicited with 40 volts of Faradic current applied through the skin (see Fig. 11). The left eye

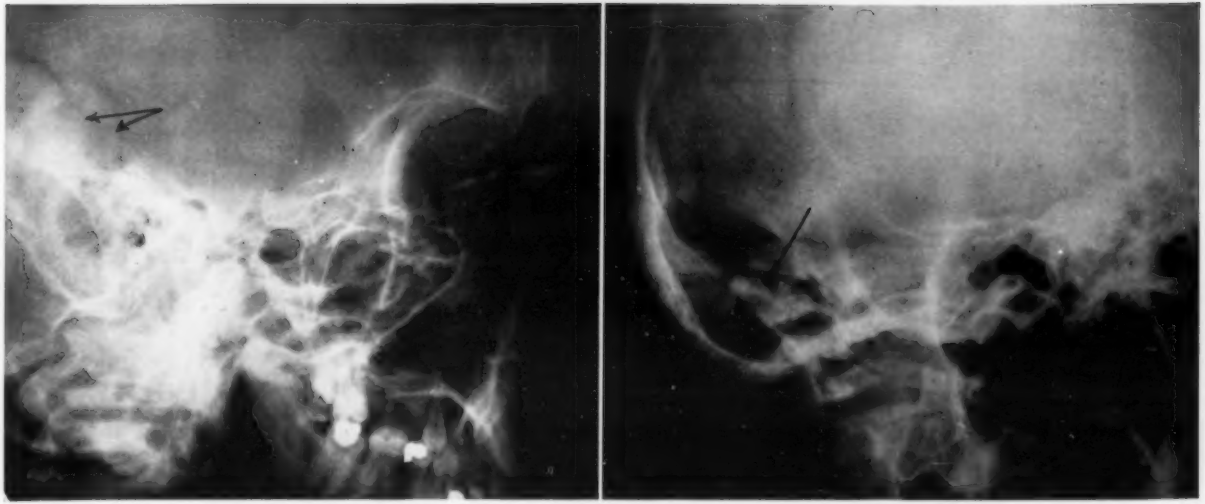

Fig. 12. Case 3. Roentgenograms demonstrating the fracture in the left parietal bone and the fracture line in the left petrous.

was moist, and the Schermer test showed lacrimation in the left eye that exceeded that in the right. There was also a loss of taste on the anterior two-thirds of the tongue on the left side. Functional tests demonstrated normal hearing on the right side, but total deafness on the left. No labyrinthine response was obtained after 40 cc. of ice water had been in-

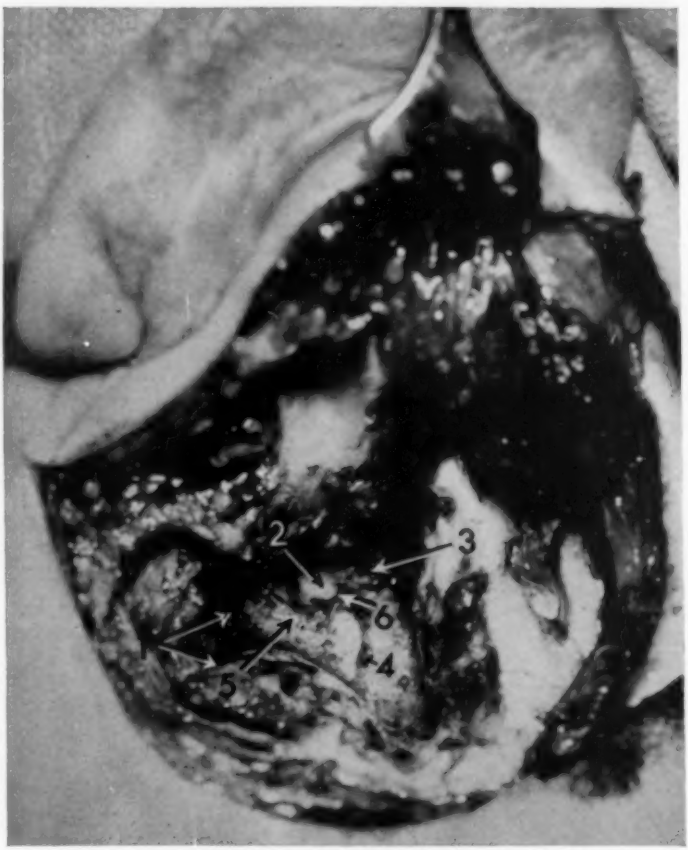

Fig. 13. Case 3. Photograph taken during operation at completion of radical mastoidectomy showing fracture through the horizontal semicircular canal.

Arrows indicate: 1. Mastoid tip; 2. Promontory; 3. Epitympanum; 4. Open fracture line across horizontal canal; 5. Exposed vertical course of facial nerve; 6. Mass of scar in region of intra tympanic portion of facial nerve.

jected into the left external canal during a period of 40 seconds. There was neither spontaneous nystagmus nor past pointing.

X-ray studies demonstrated a comminuted fracture of the left parietal bone without depression, and a fracture line which extended into the left petrous bone (see Fig. 12).

Because of the complete paralysis of the face of over five months' duration, which had been apparent immediately after the accident, and because of the marked tearing in the left eye and the deformity of the bony external auditory canal, it was felt that the facial nerve had been

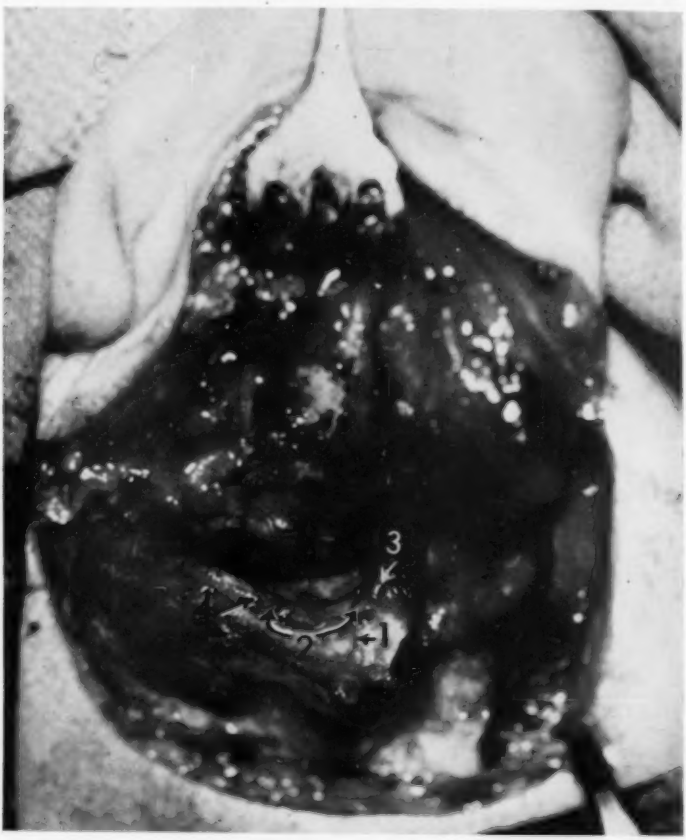

Fig. 14. Case 3. Photograph at completion of operation showing nerve graft sutured in place.

Arrows indicate: 1. Fracture site across horizontal canal; 2. Nerve graft; 3. Proximal stump of facial nerve; 4. Distal stump of facial nerve.

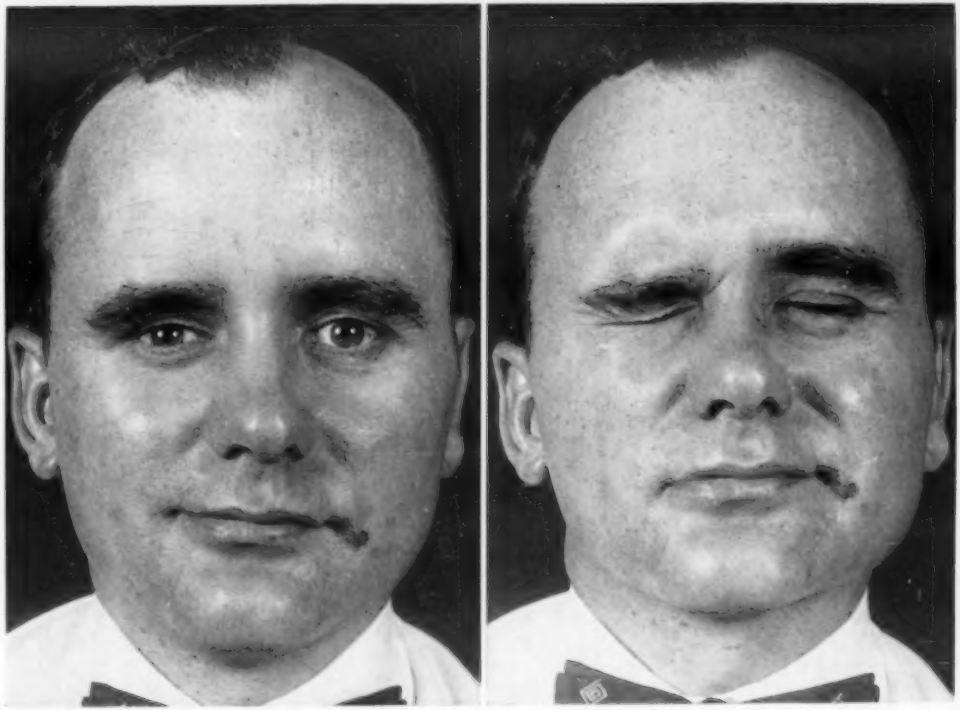

Fig. 15. Case 3. Post-operative photograph taken on March 5, 1956 (9 months after operation). Note. This photograph was added two months after presentation of this paper.

injured at a point distal to the geniculate ganglion in spite of the obvious fracture through the otic capsule, the total deafness and the loss of vestibular function.

On June 1, 1955, a left radical mastoidectomy was performed. It was found that the patient had a large pneumatic mastoid, which contained old blood but was free from any evidence of suppuration. Two widely separated fracture lines were noted in the temporal squama. The fracture had extended into the tegmen of the mastoid, where there were spicules of bone lying attached to discolored dura. As the antrum was excavated, a wide fracture line through the horizontal semi-circular canal and into the vestibule was visualized. The region of the intra-tympanic segment of the Fallopian canal was occupied by a mass of scar tissue. After completing the radical mastoidectomy, scar tissue in the region of the intra-tympanic portion of the facial nerve was removed, and it was found that the nerve had been severed just distal to the geniculate ganglion (see Fig. 13). A small stump of rather normal appearing nerve was preserved at this point, and the distal stump of the nerve was found just below its pyramidal segment. The ossicular chain had been disrupted, and all the ossicles including the displaced stapes were found in the middle ear. A 2 cm. free graft from the great auricular nerve was used to bridge the deniscence in the facial nerve (see Fig. 14). Application of this graft was difficult because of the extremely short stump at the geniculate ganglion.

The patient's postoperative convalescence was uneventful. The wound remained clean and free from evidence of suppuration.

At the time of the patient's last examination, on Jan. 3, 1956, the ear was dry. The patient stated that his face had felt slightly different during the past month and that he was having less difficulty in talking. On attempted forceful closure of the eyes, the corner of the mouth would elevate. Apparently he was experiencing the first bit of functional return since operation, which was seven months prior to this examination.

SUMMARY.

Case reports of three patients suffering from complete peripheral facial paralysis due to middle fossa fractures have been presented.

One, Case 1, had nearly normal hearing pre-operatively. The operation was a simple mastoidectomy with removal of bone fragments at the pyramidal segment of the facial nerve, and decompression of the nerve from the geniculate ganglion to the stylo-mastoid foramen. Decompression of the intra-tympanic portion of the nerve was accomplished after further elevating the displaced short crus of the incus and deepening the fossa incudis to create space through which to operate. The postoperative hearing is nearly normal. Although complete return of function of the facial muscles did not take place, there was marked improvement, which began five days after operation and had reached a fairly satisfactory level two months later.

In Case 2, the patient had multiple cranial nerve palsies and a delayed facial paralysis. The longitudinal fracture apparently extended to the region of the Gasserian ganglion. Preoperatively, there was a mixed type of deafness with 53 db loss in the speech frequencies. The operation was a modified radical mastoidectomy, and decompression of the facial nerve. Return of function of the facial muscles began about four weeks postoperatively. The patient continued to have perceptive hearing loss of 31 db in the speech frequencies, but there was considerable improvement over the preoperative level.

Case 3 was that of a patient who had a fracture through the labyrinth and cochlea. The facial nerve was severed in its intratympanic course. Repair was accomplished after debridement of the nerve ends and the use of a 2 cm. free graft from the great auricular nerve. A radical mastoidectomy was required. At the time of this writing, January 4, 1956, just seven months postoperatively, the patient is showing his first signs of return of function by his ability to move the corner of his mouth slightly. There appears to be better tone in the facial tissue, and the patient has noted less difficulty with speech during the past month. If an excellent result were to be obtained, I believe that the first signs of returning function would have appeared earlier.

It is generally recognized that the prognosis for complete, or at least functionally adequate recovery from facial paralysis due to head injury, is good in the vast majority of cases. In all probability, surgical intervention need be considered in less than 10 per cent of such patients. Such consideration must include careful and complete study to determine, insofar as possible, the severity of the nerve lesion and its surgical accessibility. In properly selected cases, the results of operation to repair damage to the facial nerve will be beneficial and gratifying. If the preoperative hearing is serviceable, every effort should be made to preserve it. A radical mastoidectomy is not necessary in every case in which operation is required.

ADDENDA.

L. M., No. 811204, the patient described in Case 3 was examined on March 5, 1956, at which time returning function was noted (see Fig. 15) and further improvement is likely.

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AN OTOLARYNGOLOGIC APPROACH TO MALIGNANT EXOPHTHALMOS.*

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It is not the purpose of this paper to deal with the etiology or pathology of progressive or malignant exophthalmos, but rather to review the historical background of the surgical relief of the condition and to bring again to the profession a rhinological approach to the problem. Progressive, or malignant, exophthalmos is a serious disorder unless controlled by medical or surgical therapy. Not only will it cause blindness, if progression goes on, but also corneal ulcer, ophthalmitis, and even meningitis and death.

The first operative procedure which I have been able to find in literature was reported by R. Foster Moore[1] in 1920. He made an incision through the whole length of the inferior conjunctival fornix, exposed the contents of the orbit, and with forceps picked away piecemeal as much of the orbital fat as possible. He estimated that a heaped-up teaspoonful was thus removed. Afterwards, the lids were easily approximated and were in part sewn together over the cornea, as also were the left lids, and by this means both eyes were preserved.

In 1930, O. Hirsch[2] reported a decompression of the orbit for progressive exophthalmos by removing the floor of the orbit, using the canine fossa approach and entering the antrum of Highmore; then removing the floor of the orbit, leaving only a narrow strip of bone containing the infraorbital nerve, thus protecting the nerve. He also states that to give more room, this bone and nerve may be removed, but loss of sensation or hypesthesia of cheek will ensue. We have never done this type of operation, but in talking to Theodore Walsh of Washington University School of Medicine, he stated that he

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did one through the canine fossa, but that it did not work, and he had to do a Sewall type decompression.

It was not until 1932 that Naffziger and Jones[3,4] reported their surgical treatment of progressive exophthalmos following thyroidectomy. At that time, they reported six persons who had been operated upon by their method. In these articles they established the principal etiological factor in this condition. The Naffziger operation is an operation performed by neurological surgeons and is described thoroughly in these articles. They were the first to explain the pathology of the condition, and their histologic sections confirm their opinion. The method they employ consists of entrance to the skull through the frontal region by means of a transverse incision from the temporal fossa of one side to the temporal fossa of the other side, going across the fronto-parietal region immediately behind the hairline. The scalp is dissected from the pericranium, and is reflected down, exposing the entire frontal bone on both sides, down to the frontal sinus. Bilateral frontal flaps are then fashioned, the hinge of each being the temporal muscle. A strip of bone is left in the center to stabilize the bone flaps when replaced. The dura is now separated, with a broad spatula, from the floor of the anterior fossa and down the sphenoidal ridge and the base of the anterior clinoid. Then the roof of the orbit is removed and the optic nerve is decompressed by removing the roof of the optic foramen. When this is finished, the orbital fascia is opened, being careful not to cut the frontal branches of the Vth nerve. The entire bony roof of the orbit is then removed. (See Fig. 1.)

In 1935, Swift[5] of Seattle, Wash., reported on Malignant Exophthalmos and Operative Approach. He reported one case on which he did a modified Kroenlein operation with good result.

In 1936, Sewall[6] read a paper before the Western Section of the *American Laryngological, Rhinological and Otological Society*, on Operative Control of Progressive Exophthalmos, and this was published in the *Archives of Otolaryngology*.

In 1939, Kistner[7] reported two cases operated upon by this method and one case by the Naffziger method.

In 1945, Schall, L. A., and Reagan, D. J.,[8] reported two cases operated upon by the Sewall method. In their conclusions they state, "Two cases of malignant exophthalmos are presented that were treated by decompression into the sinuses following the method proposed by Sewall and first used by

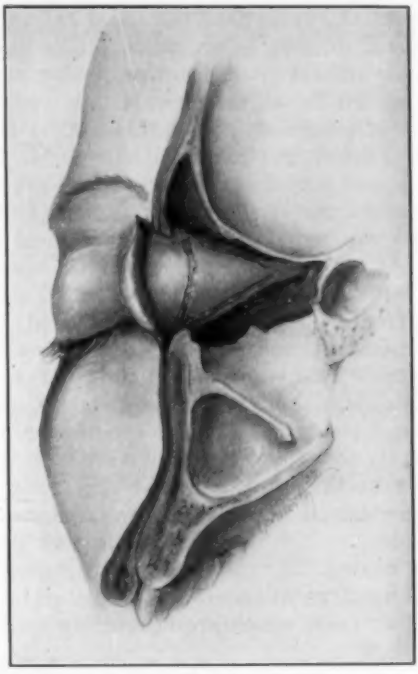

Fig. 1.

Kistner. We agree with Kistner's conclusions that this operation is:

1. Less disfiguring and formidable than the Naffziger operation.

2. An actual rather than a potential space is created.

3. Sometimes the roof of the orbit is occupied by extension of air cells which the Naffziger technique avoids entering.

4. There is no subjective or objective evidence of pulsation

in the orbital contents which may follow the intracranial operation."

In the Sewall[6] type of operation, we use the fronto-ethmoid technique which was described by him in November, 1926, in the *Archives of Otolaryngology*. Anyone who has done this type of operation and removed the lamina papyracea has seen the orbital contents bulge into the nasal cavity, and this is especially true if during the operation a tear has been made into the capsule and fat of the orbit protrudes. It is this fact, I am sure, that led Sewall to advocate this type of operation for the relief of progressive exophthalmos. It is possible in this type of operation to remove the three walls of the orbit; also, if need be, one may decompress the optic nerve by removing the lateral margin of the optic foramen. In this type of procedure, one can remove supraorbital ethmoid cells if they are present. After the lamina papyracea has been thoroughly removed, at least as far back as the sphenoid sinus, and the floor of the frontal sinus is entirely removed, the ethmoid labyrinth exenterated and the lining of the frontal removed, incisions are made by opening into the orbital fascia. Kistner[7] described three incisions: two anteroposterior incisions, and one transverse incision. The anterior posterior incisions are placed, one at the lower border of the resected lamina papyracea, the other at the upper border. The transverse cut is started at the external limit of the frontal sinus and carried internally across the anterior ends of the two previous incisions. Before closing, the mucoperiosteal flap should be placed to protect the nasofrontal opening as described by Sewall.[9]

We report two cases which are interesting and instructive.

Case 1: W. H. D., male, age 31, truck driver: First seen June 24, 1947, and stated the proptosis was first noticed two months previously. Weight loss eight pounds past six months; appetite very good. No tremor noted except when hungry. Examination showed marked exophthalmos of both eyes, but right eye worse, inability to close lids, showed the stare peculiar to exophthalmos. Proptosis 24 mm. right eye, 21 mm. left eye, October 10, 1947. There never was any enlargement of right or left lobes of thyroid, or of the isthmus. No substernal thyroid. Laboratory findings all negative except basal metabolic rate which went to +44. Diagnosis: Pituitary-thyroid progressive exophthalmos. Large corneal ulcer on left. Left side decompression done November 6, 1947; right side decompression done November 20, 1947. Uneventful recovery; well to present time.

Case 2: Mrs. E. W. Mc., age 60. This white female entered Emanuel Hospital August 25, 1953, after having been released from the hospital one month previously, at which time a thyroidectomy was done. Since discharge, she had had a persistent pain in both eyes, headaches over eyes, and especially the left. Exophthalmos progressively getting

worse. Physical examination showed blood pressure 146/96; eyelids were sutured together and bandaged. Evidence of thyroidectomy scar. Eyes checked by Dr. U'Ren, an ophthalmologist. The diagnosis of progressive malignant exophthalmos was made, with intense retro-orbital and periorbital edema. Both corneas were ulcerated, particularly the left. On August 27 a diagnosis was made of left endophthalmitis with eyeball full of pus. On August 29 an enucleation of left eye was done. The patient was referred to us on September 29, 1953, with a recommendation for a decompression operation of right eye. On examination, we found a deep ulceration of about half of the cornea. On October 3, 1953, a decompression operation was done on the right eye. When through with the operation, she could close her lids. On the sixth day the ulcer was healed. No doubt the left eye could have been saved if seen sooner. Her vision is poor, due to the corneal scarring.

In conclusion, we advise an early decompression for progressive exophthalmos in order to save eyesight and possible death from meningitis.

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ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from October 1 through 7, 1956. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck, and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

APC VIRUSES IN RESPIRATORY DISEASES— CLINICAL ASPECTS.*

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Respiratory diseases of apparently non-bacterial origin have been a perennial problem to those dealing with the illnesses of children, of military recruits and of industrial workers.

May I review with you, certain recent advances in our knowledge of these diseases, mainly the work and experience of Drs. Rowe, Huebner and Bell, at the National Institute of Health, and Dr. Ward, at Johns Hopkins.

The story as I will review it,‡ in a sense, had its beginning with physicians in your field. In 1952 and 1953, tonsillar and adenoid tissues were collected from children undergoing surgery at the Children's Hospital in Washington, and at several other hospitals. These tissues were minced up and placed in test tubes for tissue culture in the hope that they would grow *in vitro* and provide a host for the culture of common respiratory viral agents. Instead, over 50 per cent of the tissues themselves were found to contain viral agents which could then be transmitted serially through other tissue cultures.[1] These viruses and a group of closely related agents are now named the adenoidal - pharyngeal - conjunctival viruses for which the abbreviation APC is used. At about the same time Hilleman isolated, from cases of acute respiratory disease in military personnel, a virus called RI-67, which shares the characteristics of the APC group.[2] This group of APC viruses includes the cytopathogenic agents which were originally isolated from tonsils and adenoids and have subsequently been

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‡ Parts of this talk have been published previously in Clinical Proceedings of Children's Hospital, Washington, D. C., 11:152-158, 1955.

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found in conjunctival, pharyngeal and fecal material.[3] The human sources of these viruses indicate why the name was proposed: they were first isolated from adenoid tissue; human conjunctival and pharyngeal material have been the major sources of these viruses subsequently. These agents are not pathogenic for laboratory animals. They do show a unique destructive effect in tissue culture of epithelial tissues, particularly of the serially propagated HeLa cell strain of human cervical carcinoma. At least some of the strains cause similar effects in monkey kidney and in human embryo fibroblast in tissue culture. The viruses are ether resistant, heat labile and filterable. They resist antibiotics. Members of the group share a group specific soluble antigen which gives a group reaction in a complement fixation test. This means that by a complement-fixation test these types cannot be differentiated. The agents do produce a type specific neutralizing antibody and on the basis of neutralization tests it can be said that there are at least 10 or 11 immunologically distinct types of APC viruses.[4]

Serological studies, mainly neutralization tests, indicate that over 50 per cent of infants in the 6- to 11-month age group show evidence of having been infected with at least one type of APC virus.[5] There is a sharp rise in evidence of infection from ages one to two. There is a less prominent increase in the number of individuals with neutralizing antibodies from ages three to five. Gradually thereafter, there is increasing evidence of individuals having been infected with at least one of the virus strains.

What do these viruses do clinically? Sufficient data are not available yet concerning the clinical aspects of most of the APC virus types. Two well studied outbreaks of an illness for which the name pharyngo-conjunctival fever is proposed, have provided reasonably adequate information concerning the etiological associations and the epidemiology of Type 3 APC infection.[5,6] Evidence that pharyngo-conjunctival fever is etiologically related to Type 3 APC viruses, includes the facts derived from an epidemiological study, that: *1.* These agents invaded the tissues of persons with this clinical entity; in other words, the individuals showed evidence of antibody response; *2.* they were found in the site of the pathologic le-

sion in the clinical entity, *i.e.*, found in the throat, if the throat was infected; in the eye, if the eye was infected; *3.* they were not present in well persons who were in intimate contact with the individuals who had the illness; *4.* the viruses were present in the acute stage of illness, not before and not after; *5.* they were associated consistently with a particular clinical entity; and *6.* they were not found in other illnesses occurring at the same time as the outbreak studied or in sporadic studies of many cases of respiratory and other kinds of illnesses.[6]

In addition, recent inoculation of these agents in human volunteers has reproduced essentially the clinical entity which I will describe.[7] It is interesting to observe that originally, the inoculation of these agents into the nasopharynx did not produce clinical illness, but when the material was inoculated into the conjunctiva, the characteristic illness was reproduced.

The first outbreak of Type 3 APC virus infection associated with a clinical illness (pharyno-conjunctival fever) occurred in a hospital ward and included eight patients. In these patients, pharyngitis was a common complaint and finding. Two patients had exudative pharyngitis. Rhinitis was a very frequent symptom and sign. High fever lasted for five to six days. In six of the patients, cervical lymphadenopathy and conjunctival erythema were noted.

One youngster in this outbreak may be interesting to your group, particularly as she had a complicating sinusitis and otitis. Most of the other individuals with this infection whom we have seen during the summer months do not have these complicating features. The patients whom we have seen during the winter months seem more likely to have paranasal obstructive disease.

There was lack of severe leukocytosis and toward the end of the illness, there was relative leukopenia, with a slight lymphocytic predominance.

Table I summarizes the symptoms and signs that occurred in a much larger group of individuals in a summer day camp outbreak of this disease.[6,9] The symptoms included fever, complaint of sore throat, red eyes, headache, malaise, and occasionally aching and a general feeling of malaise which was

TABLE I.

MAJOR FEATURES OF PHARYNGO-CONJUNCTIVAL FEVER IN A CAMP OUTBREAK.

SYMPTOMS	Frequency
Fever	90%
Sore Throat	70%
Red Eyes	66%
Headache	68%
Malaise, Weakness	72%
SIGNS	**Frequency**
Fever 102°-104° for 4-5 Days	90%
Pharyngitis	75%
Cervical Adenopathy	75%
Posterior	66%
Rhinitis	50%
Conjunctivitis	75%
Unilateral	25%
Bilateral	50%

more common in older people, such as the parents of the children involved. Cough was not really a prominent symptom. Vomiting, diarrhea and epistaxis occurred in a few of the patients. High fever, lasting four or five days, occurred in almost all the patients. Pharyngitis consisted predominantly of hyperplasia and erythema of lymphoid tissue on the posterior pharynx and on the anterior pillars of the tonsillar fauces. Lymphadenopathy was distributed most strikingly in the posterior cervical areas. Rhinitis was manifest by rhinorrhea and by mild inflammation. Conjunctivitis affected one or both eyes; in some instances the conjunctivitis was unilateral initially, and became bilateral. Both the bulbar and palpebral conjunctiva were affected with superficial inflammation that was manifest mainly by injection of vessels. There was no pruritis, but there was a great deal of watery exudation. A few patients showed signs of meningismus. A few had cough, and ronchi could be detected.

The four major clinical features, then, of pharyngo-conjunctival fever are fever, conjunctivitis, pharyngitis, and posterior cervical lymphadenopathy. There may be several combinations of these findings in different patients.

Pharyngo-conjunctival fever occurs epidemically and sporadically.[6] Epidemicity is probably defined by the degree of

immunity existing in the community or group attacked. It affects all ages, all sexes, although mostly children again, because of their relative susceptibility. Among the adult contacts the mothers were more frequently infected than the fathers. This probably also represents a matter of opportunity to have been infected previously. Virus is spread by direct contact almost surely, and probably by respiratory secretions.

The usual portal of entry is somewhat in question because of the points brought up by the human volunteer experiments. It is hard to know what is happening in natural infection if in an experimental infection one cannot induce the disease by nasopharyngeal inoculation. There is a very high attack rate among susceptible individuals; in children four to seven years of age, for example, the attack rate was 72 per cent in the summer day camp. There is a low or absent carrier rate; no individual who did not have the clinical syndrome showed evidence of infection with the virus. There is evidence of advancing immunity with age. The incubation period is five to six days. The period of communicability is up to nine days. That swimming pools play a role in the spread of this disease is suggested by the several instances in which outbreaks occurred among persons having contact with camp or neighborhood pools.[6,10,11] Demonstration of virus in the water has not been possible. Two possible explanations for the apparent association of pharyngo-conjunctival fever with pools come to mind: *1.* the pools are a congregating place during the summer and congregation facilitates direct spread; *2.* swimming in pool water causes conjunctival irritation and allows ready ingress for virus spread by contact with other persons.

Table II summarizes what we presently know about the sources of the other APC virus types. It may be of particular interest to your group to know that all of the Types 1 through 6 have been isolated at one time or another from tonsillar and adenoid tissue. Types 1, 2, and 5, have been found frequently, not only by our group but also by other laboratories, in pharyngeal lymphoid tissue removed at surgery.[12] The question of what the viruses may be doing in those tissues is one for which I think we have no real answer. One hypothesis is that persons undergoing acute infection with these agents

TABLE II.

RESUME OF HUMAN SOURCES OF APC VIRUSES.

APC TYPE	1, 2, 5	3	4	6	7	8	9	10
Tonsils, Adenoids	+	+	+	+				
Febrile Pharyngitis	+	+						
Pharyngo-Conjunctival Fever		+						
Acute Respiratory Disease			+		+			
Kerato Conjunctivitis						+		
Catarrhal Conjunctivitis				+				+
Poliomyelitis Survey							+	

carry the viruses at least for periods of time, in the pharyngeal lymphoid tissue. In partial support of this hypothesis is the fact that in the Washington, D. C., area, for example, Type 3 virus had never been isolated from a tonsil or adenoid until the year when there was a widespread outbreak and widespread occurrence of Type 3 in acute disease. The next year a few tonsils and adenoids had Type 3 in them.

Types 1, 2 and 5 have also been found in less specific febrile pharyngitis in children in studies which were not so well controlled clinically as those I have mentioned.[2,13] Type 3 has also occurred in a few cases of febrile pharyngitis,[3,14] and in a disease resembling roseola infantum.[15] Type 4 virus, known also as Respiratory Illness 67, or RI-67 virus by Hilleman, has been found as "recruit disease," what the Commission on Acute Respiratory Disease has called ARD, and in primary atypical pneumonia-like illness almost exclusively in military recruits, Army and Navy.[2,16,17,18,19] Type 6 has been found in two patients with catarrhal-conjunctival infection and mild respiratory symptoms.[10] Type 7 has been found in distribution similar to Type 4.[14] Type 8 has been found in cases of epidemic keratoconjunctivitis in industrial workers.[21] Type 10 has been found in a case of catarrhal conjunctivitis,[4] Types 9 and 11 were isolated from feces in a survey of patients with poliomyelitis-like disease.[4,22]

There remain many problems in this field. First of all, are similar groups of viral agents responsible for many other

respiratory diseases so often seen in practice? Second, concerning this very prevalent group of APC viruses, there are many questions which one might ask, for example, what are they doing in the tonsils and adenoids? Could they be associated with the process of adenoid and tonsillar enlargement? What other clinical entities might be associated with APC virus infection? What therapy would be indicated for infection with these agents? In our experience no antibiotic or chemotherapeutic agent seemed to have a therapeutic effect in the patients studied for the acute uncomplicated illness. Those few children who had complicating otitis or sinusitis did seem to respond to antibiotic therapy; it was presumed in these cases that obstruction of the paranasal orifices allowed bacterial infection to occur.

Another most provocative question is: What are the possibilities of immunizing human beings against these agents? The viruses are very prevalent. There is evidence that they are responsible for a large amount of respiratory disease. In volunteers, some vaccinated individuals resist challenge with the type virus in the vaccine.[23] The agents can be grown readily in tissue culture, and a vaccine has been made and could be made on a larger scale. It is not too unlikely that an effective vaccine might be developed which would eliminate a significant amount of respiratory disease.

In summary the APC, or adenoidal-pharyngeal-conjunctival viruses, are newly isolated epitheliotropic tissue culture agents. They have been associated predominantly with an illness named pharyngo-conjunctival fever and with respiratory disease in military recruits. If the major features of pharyngo-conjunctival fever, conjunctivitis, pharyngitis, and cervical lymphadenopathy are present, or a combination of them is present in the face of an epidemic, I think one can be fairly definite about the clinical diagnosis of Type 3 APC infection without having a virological diagnosis made.

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SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M.D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

A NEW METHOD OF BRONCHOSCOPY WITH CINEMATOGRAPHY AND PHOTOGRAPHY.*

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ROGER J. EDWARDS, M.D.,

and

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Reims, France.

A new French apparatus now permits an endobronchoscopic film to be produced in the course of each bronchoscopy. An objective record of the specialist's examination may thus be kept, a document which may be studied or discussed in the interest of the patient.

It would not occur to a radiologist to be content with a fluoroscopic examination when he detects a pathological condition of the lungs. The endoscopist, likewise, has felt for many years that his reported observations, often recorded in considerable haste and illustrated with a diagram (even when such a diagram is drawn with the greatest skill and accuracy), represents records which are still very incomplete.

Dr. Paul Holinger, of Chicago,[1,8,9] should be credited with having produced the first endobronchoscopic films. His films are known not only in America but throughout the world, as well. With his photographic equipment, endobronchoscopic 16 mm. color films of rare quality may be made at 16 or 24 frames per second. In France, Dr. André Soulas has used similar equipment, obtained in Chicago nearly 10 years ago, to produce the first European endobronchoscopic films.[10,11]

As a result of improvements recently achieved with a new bronchoscope developed by Fourestier, Gladu and Vulmière, accurate records may now be obtained through post-telescopic cinematography, at 16 frames per second, utilizing 8 mm.

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*Travail du Centre Régional de Lutte contre le Cancer (Directeur: Professeur Raymond Lefèvre), et du Service de Pneumo-Phtisiologie de l'Hôpital de Reims (Chef: Pierre-M. Dubois de Montreynaud, M.D.).

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films, which are not expensive, and recently 16 mm. films have become possible as well.[2,3]

The apparatus, of the usual diameter, is easily handled. For this reason, we have been able to do systematic cinematography in our service since June, 1954. Every bronchoscopic examination thus becomes the object for a film, yielding valuable objective data, which may later be studied at leisure and compared with the rest of the patient's record. This method gives a faithful reproduction of the bronchi, whether normal or pathological, and is applied in several Paris hospitals, especially in the Laënnec Hospital (Pr. Et. Bernard), in several province hospitals and particularly in the Reims hospital.

The bronchologist very quickly discovers the secrets which allow him to obtain excellent films. When the operator and personnel are accustomed to the technique, filming the examination requires no more time than a normal bronchoscopy; it permits excellent illumination during the bronchoscopy, and there is no delay in seeing the photographs, which can be inspected as often as desired once the film is developed.

The life-like character of the endobronchoscopic pictures is particularly interesting. The normal movements of the bronchi are: inspiratory enlargement of the bronchial caliber, ascending expiratory movements of the whole bronchial tree, transmission of heart beats, suppleness of the mucous membrane; also important changes observed in asthma, cancer, etc., or in experimental studies in animals, can be easily followed.

By this method the head of a large hospital service may note the state of patients' bronchi. Formerly this was possible only for the bronchoscopist. The same is true for assistants and students; the educational nature of these films cannot be too strongly stressed.

At the time of the medical-surgical conference, in an era when pulmonary or bronchial resection is essentially conservative, it is particularly important for the bronchoscopist or surgeon to have information which will be useful at the time of intervention.

Systematic endobronchoscopic cinematography will hence-

forth permit each patient to have documented observations which are actually complete. It is easy for the bronchologist to have a small projection room near his office where a projector is permanently installed. It takes no longer to run a film than to view a series of tomograms. Such endobronchoscopic documents have considerable interest, not only for the bronchologists, but also when presenting their work before professional societies, bronchial lesions can be shown as well as discussed.

DESCRIPTION OF THE APPARATUS.

It was necessary to find a simple means, available to every practitioner to combine classical bronchoscopy and photography in a simple operation, just as it is possible to pass from radioscopy to radiography within a few seconds, through the simple adjustment of a cassette.

Today the problem is solved, and we have an apparatus utilizing bronchoscopic tubes of the usual diameter, and the usual telescopes through which the pictures are taken, with illumination 200 times more powerful. Photography or cinematographic recording in every interesting detail may be made instantaneously with the simple aid of a photographic apparatus, or with any commercial camera desired, placed against the eye piece of the telescope.[6,7]

This apparatus is composed of several parts (see Figs. 1 and 2):

The bronchoscopic tube; a particular lighting indispensable for photographs or cinema, including a powerful light source and a conductor for that light, with an increased lighting coefficient.

Direct and lateral vision telescopes adapted to the Wolde bronchoscope and provided with an anti-vapor system, the importance of which is paramount in the success of photographs or films.

A number of parts, including an adjustable electric supply, an aspirator for cooling the lamp and a compressor for the anti-fogging system, all of which are grouped according to the models.

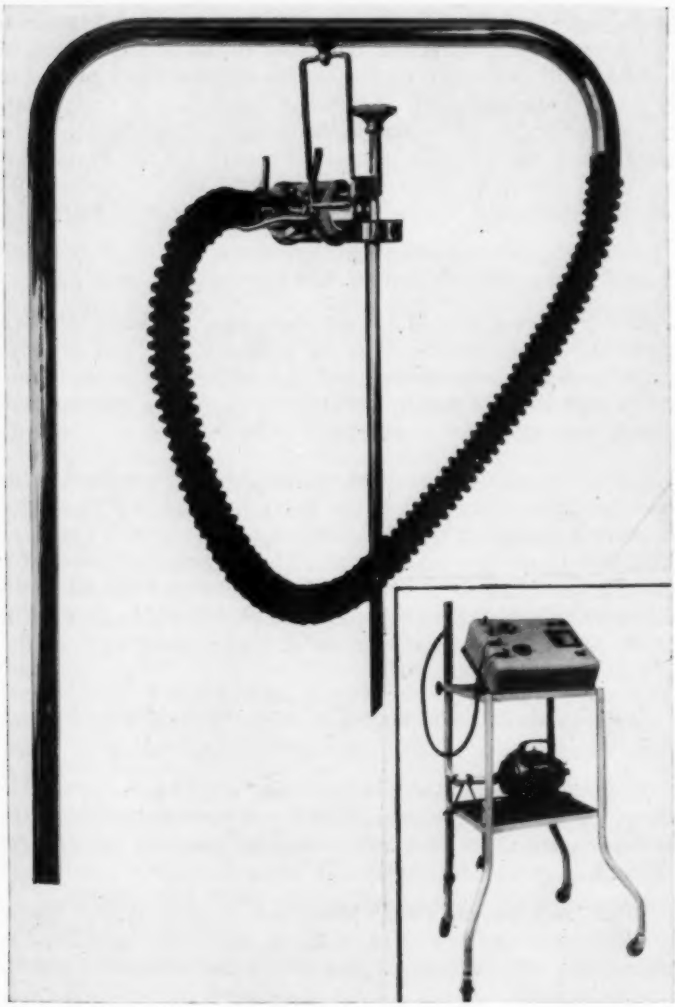

Fig. 1. Photography of the endoscope from Fourestier, Gladu and Vulmiere.

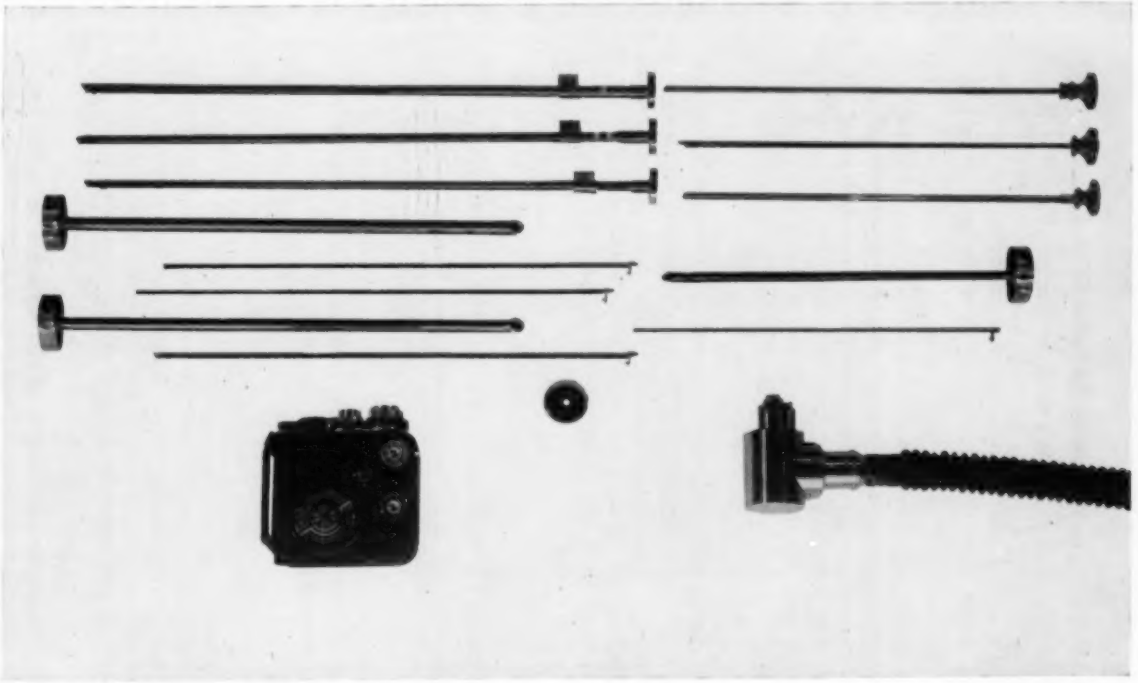

Fig. 2. Details of the equipment: A. on the left, for adults, two tubes, one direct telescope, two right-angle telescopes, two direct quartz-rods, one lateral quartz-rod; B. on the right, for children, one tube, one quartz-rod and three telescopes; C. below, the motion-picture camera, the adapter and the housing which contains the bulb.

THE BRONCHOSCOPIC TUBE.

The bronchoscopic tubes present few modifications in relation to those of classical instrumentation.

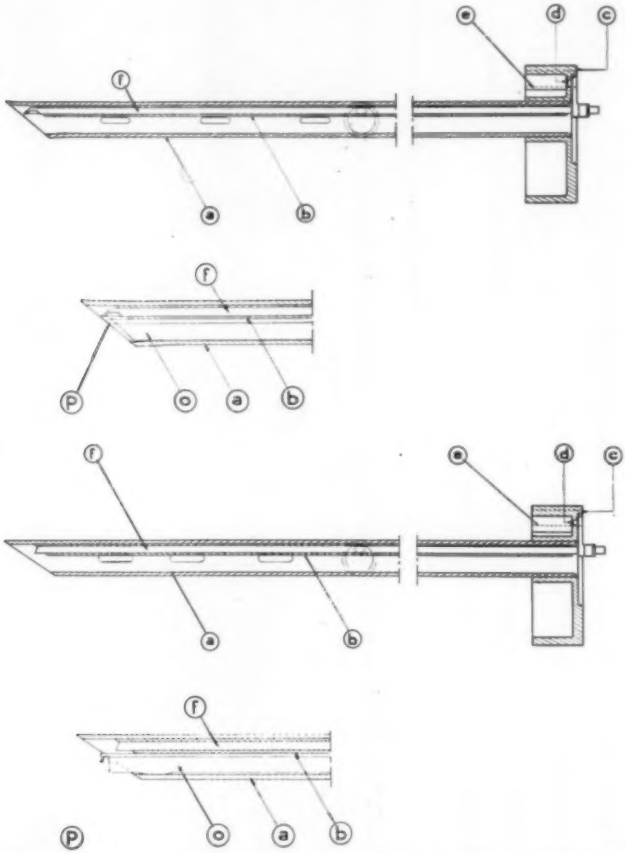

Fig. 3. A. the bronchoscopic tube; B. the light carrier-canal for the quartz-rod; F. the quartz rod; O. the telescope (direct or right angle).

The diameter and length of the tube for an adult are exactly the same as those currently in use *i.e.*, 8 mm. diameter and 40 cm. in length; the thickness of its wall is 0.5 mm.[4]

The tubes must be made of an alloy with a nickel base instead of brass, so as to be perfectly rigid to avoid breaking the quartz rod, in the course of bronchoscopic maneuvers. This provides a much greater resistance to bending, which is very important if the larynx is anterior or the neck short (see Fig. 3).

The classical light carrier canal into which the quartz rod is introduced is soldered to the interior of the principal tube, so that the bronchoscope may be rotated in the bronchus without trauma. The light carrier canal has a diameter of 3 mm. and the wall is 0.1 mm. thick. The interior of the tube is covered with an electrolyte deposit of black, flat, non-reflecting nickel.

The distal extremity of the tube has a classical beveled aspect, but at its proximal extremity, a plate 50 mm. in diameter, permits the illuminator, Galileo magnifier or proximal telescope, to be adjusted or removed rapidly by means of pegs. We now have tubes for children, based on analogous principles.

LIGHTING.

The lighting constitutes the true originality of the system. Until recently in France a distal light source has been used. The illumination given by this small bulb, a masterpiece of manufacturing nevertheless, is not comparable to that obtained with the new apparatus. A proximal light source is now used, the size and power of which is limited only by optical considerations. The image of the filament is transmitted to the distal end of the scope by a transparent rod of fused silicon (quartz-rod).

a. The light source is composed of a standard microscope-type lamp of 6 or 8 volts, 5 amperes, with a 2 mm. filament. This is placed in a metal case of a size which permits ease in manipulation. The lamp housing is a cylinder 90 mm. long and 50 mm. in diameter. Its weight is about 650 gr. It is cooled by air drawn through the lamp housing (see Figs. 4, 5 and 6).

The filament is centered in the housing in such a manner that it is at the optical center of a concave mirror. The direct

and reflected light is then picked up by a condenser composed of two convex plane lenses with aspheric surfaces. The beams are then directed at a 90° angle by a prism to the exit of the housing, which consists of a small orifice 3 mm. in diameter.

Fig. 4. The housing is open.

This orifice fits exactly against the proximal end of the quartz rod. When the lamp is illuminated, a special viewer permits an exact centering of the filament. The centering can be modi-

fied with the aid of three special screws which control the support of the bulb.

The lamp housing is cooled by air drawn through it. The air penetrates through 24 small holes in the bottom of the housing and escapes through a large tube on the side of the

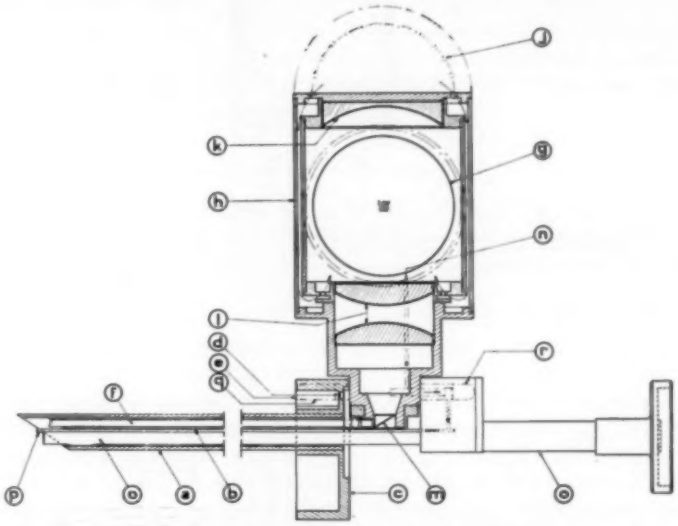

Fig. 5. The diagram shows the details of the apparatus: A. the bronchoscopic tube; B. the light carrier-canal for the quartz-rod; F. the quartz-rod; G. the bulb; H. the housing; J. the small holes through which the air is drawing for cooling; K. the concave mirror; L. the two convex plane lenses; M. the prism; N. the air pressure for anti-fogging; O. the telescope; P. the way out for the air blowing on the distal lens surface.

housing connected to an aspirator. The tube also contains electric wires for the entry of the current, and a small tube for compressed air for the anti-fogging system of the telescope. This large flexible tube, containing the air pressure tubing and the electrical wiring, leads to the aspirator-compressor, transformer and voltmeter.

The lamp housing is attached to the plate of the bronchoscopic tube by two pegs, which assure the optical accuracy of the complete apparatus, and by a supplementary peg which conducts the compressed air for the anti-fogging device of the telescope.

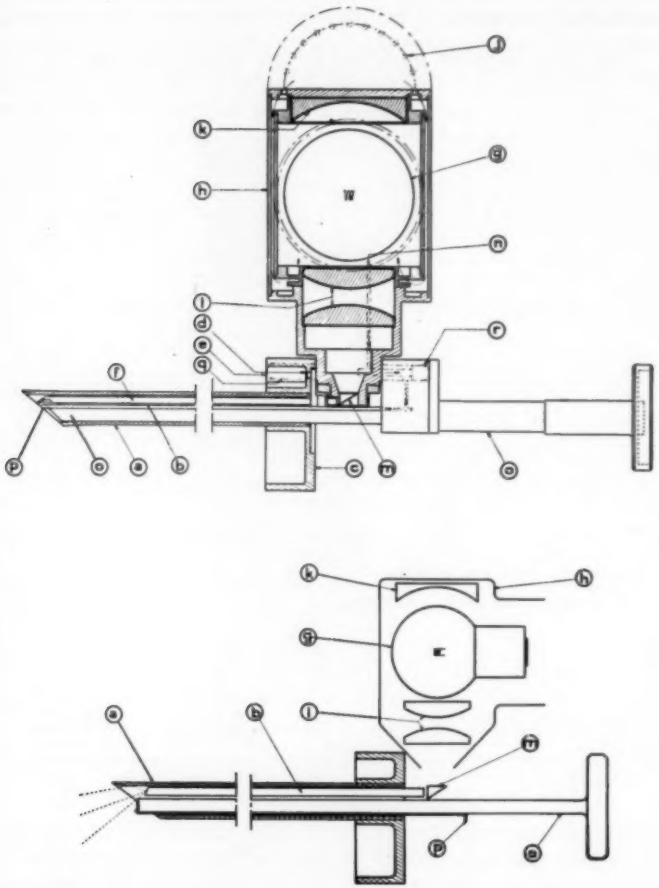

Fig. 6. The same diagram with right angle quartz-rod: A. the bronchoscopic tube; B. the light carrier-canal for the quartz-rod; G. the bulb; H. the housing; K. the concave mirror; L. the two convex plane lenses; M. the prism; O. the telescope; P. the small canal for air pressure for anti-fogging.

Simplified diagram: A. the bronchoscopic tube; B. the quartz-rod; G. the bulb; H. the housing; K. the concave mirror; L. the lenses; M. the prism; O. the telescope.

b. The quartz rod has a diameter of 2.5 mm. and is 410 mm. long. It is especially made, partially encased in silver and copper and surrounded by a protecting envelope of nickel. Its two extremities are smooth; the proximal end is cut at right angles, the distal end has a form which varies with the direction of the light tract. The rod is either beveled, to send the ray solely into the field of the direct telescope, or it terminates in a mirror which permits reflection of light obliquely, to light the field of the lateral telescopes. At the exit of the case the rod transmits 80 per cent of the light.

THE TELESCOPES.

The telescopes are constructed according to the same principle as those ordinarily sold, using the best available coated lenses; however, along the length of the telescope and in its metal casing is incorporated a very fine tube which will conduct air under pressure, which constitutes the anti-fogging system. The air pressure for this purpose is carried through the large flexible tube, described above, and passes through the peg which keeps the telescope in place. This is important, because the distal extremity of the telescope must be fixed in relation to the distal extremity of the quartz rod. After traveling the length of the telescope, the air is blown across the distal lens surface, thus eliminating fogging. This avoids the use of anti-vapor liquids which distort the photographic image.

The direct telescope is the most commonly used; however, to obtain good records it may be necessary to resort to various telescopes whose sighting axes vary from 30° to 120° with the axis of the direct telescope.

The special quartz for lateral lighting is indispensable. In the course of bronchoscopy it is easy to change the quartz in order to have the type best adapted to the telescope being used.

The apparatus which has just been described must be especially made, and certain of the parts have been patented by the National Center of Scientific Research. The material to be described next is in current use; its assembly alone is original. We shall consider these complementary parts and their mode of attachment to the bronchoscope.

The transformer furnished with a voltmeter and regulator

adaptable for 110 or 220 volts, permits a voltage of 4-12 volts. During bronchoscopy, a voltage of four to five is used. During the photography and developing of films, it is necessary to increase the voltage to 10-11 volts. A hand or pedal rheostat makes it possible to pass from one voltage to the other.

The aspirator for drawing air through the lamp housing is one-quarter horsepower.

The compressor used for the anti-fogging flow of air is of low power.

These three parts of the apparatus together may be adjusted on an instrument table, or otherwise contained in a carrying case. In either case, a metal tube will permit the electric wires and air tubes to be grouped together, and may serve as a support for the bronchoscope. A flexible tube, about 1 meter long, and containing electric wires and the small tube of the compressor, connects the metal supporting tube to the lamp housing.

Apparatus for Cine Photography: An 8 mm. camera is used with a 12.5 mm., f. 1.9 lens, at 16 frames per second.[3] At this time an 8 mm. reflex camera permitting control while the pictures are being taken is not available, but experience shows that excellent films may be made despite this fact. A 16 mm. camera with a focal range of 17 or 20, aperture f. 1.5, 16 frames per second may also be used.[2]

The still picture photographic apparatus should be a reflex type, 24x36, focal range of 35-38 mm., aperture, 3.4. The setting should be on 0.50 m. to 0.60 m.

Camera or photographic apparatus are applied by simple contact of the camera lens to the eyepiece of the telescope, a small adapter on the camera lens will insure the alignment of the contact. This adapter will contain an additional lens, to be used with a camera that cannot be focused directly.

Optional accessories:

The Galileo Magnifier: This proximal telescope may be adapted to the bronchoscope instead of the usual bronchoscopic telescope. The most diverse bronchoscopic maneuvers are made easier by magnifying the image three times. It may be used during the introduction of the tube, the examination

of the bronchi, or for obtaining specimens for laboratory examination.

The View-finder With Two Telescopes: This is composed of two Galileo view-finders, with an Eimage aspirator, permitting two simultaneous observations. It also permits observation of progress of the bronchoscopy and, therefore, is of great didactic value.

Such is the bronchoscope in its present form. To us it seems excellent. Perpetual alterations and numerous improvements to eliminate the minor disadvantages have been promised. It is essentially practical; is light, strong, but not cumbersome.

TECHNIQUE OF BRONCHOSCOPY.

We request our patients to fast the morning of the examination, to take a barbiturate on retiring the night before, and upon awakening in the morning, and we give them ½ or 1 mm. Atropine sub-cutaneously one hour before the examination. We never employ premedication with an opiate base, and only exceptionally give a curare derivative. A local anesthetic is given in classical fashion, with 5-10 cc. of tetracaine (or pentocaine) 1 per cent, with the spray and laryngeal syringe.

At the time of the bronchoscopy, the bronchoscopic tube with Jackson's light carrier may be passed through the pharynx and larynx. We prefer not to use the quartz rod lighting system during the introduction of the scope; the introduction may be difficult, and while there is some risk of breaking the quartz rod, this incident has been exceptional with us. It is important that no secretions be permitted to collect on the distal end of the bronchoscope, since such a deposit on the extremity of the rod would be difficult to remove and would prevent good light transmission; likewise before introducing the tube into the trachea or bronchi, we always check to see that no secretions are present there. It is necessary to aspirate them before the extremity of the rod or telescope becomes soiled. The success of the film depends on small details of this kind.

After these precautions, progress into the two sides of the bronchial tree is easily made. The direct telescope permits the

view of a field, which is quite extensive in length and depth. It is frequently possible to descend into the terminal branches of the inferior lobe. In the course of this observation of the

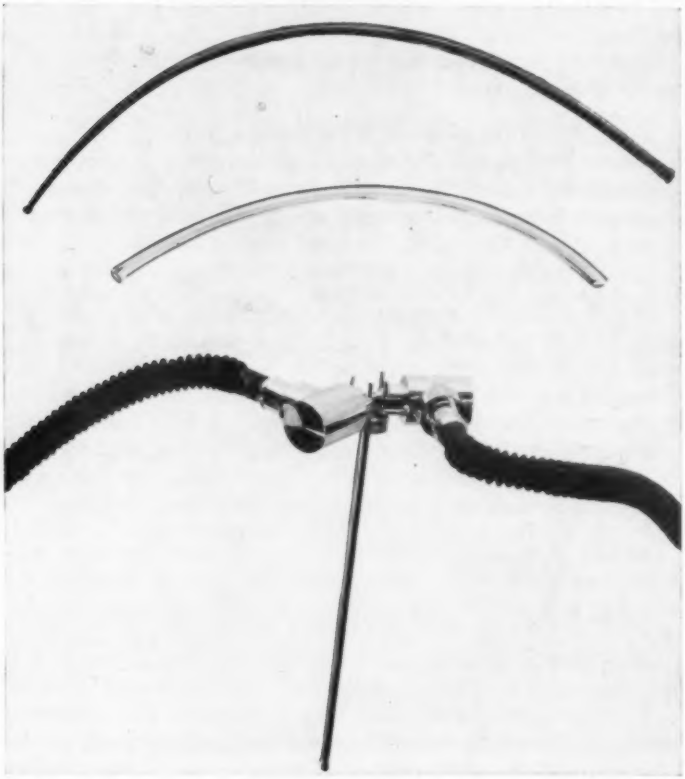

Fig. B-5. The esophagoscope: the mandril, the cover tube made of transparent plastic, the esophageal tube with double lighting.

bronchi, it is very easy to alternate the camera and the eye, and even without a view-finder it is possible to obtain very good continuous photographs, as the scope is advanced or withdrawn, which show the character of the bronchial mucous membrane. The examination of the lateral orifices is made by changing the quartz rods and telescopes. All in all, one bronchoscopy with the taking of a film does not require any more

time than an ordinary bronchoscopy; moreover, all the common endobronchic maneuvers are possible with this bronchoscope. They are performed comfortably, under very powerful lighting, which is particularly desirable if there are abundant or bloody secretions.

Cinématographic Films: 8 mm. Kodachrome "A" films at 16 frames per second are used, black and white films lacking interest. An amateur's ordinary camera is used, its only requirement being a focal range of 12.5 mm. and an aperture of 1.9. The focus is set for 0.50 m. If the camera is set for infinity, an additional lens becomes necessary. The quality of the film for clarity of image, as well as for the color quality, is really excellent. Details which were unsuspected during the bronchoscopic examination alone are evident in these films.

The bronchoscopy is completed by cinematographic study of the film on the screen. For each patient, according to the importance of the examination, one to four meters of film are taken. We believe that a film of the whole bronchial tree should be made systematically during all routine bronchoscopy. Normally, however, the photography should not prolong the examination. On the contrary the film permits it to be done in less time, since the operator knows that he can review any particular region at his leisure when the film is later projected; 8 mm. film can be enlarged to 16 mm. size for projection in large rooms, permitting the original to be kept intact. The direct use of 16 mm. films is possible, but it is intended that they be used only for teaching, or scientific publications (16 mm. Kodachrome "A" films, 16 frames per second, focal range of 17 or 20 mm., aperture of 1.5).

Finally, color slides may be made from the 8 or 16 mm. on the 24x36 mm. stock (the image then has a diameter of 10 or 15 mm.), and from these slides and enlarged color prints may be made. The diameter of the image on paper is then 75 mm.

Photography: Color photographs may be made with Ektachrome film, with a setting time of one-tenth to one-twentieth of a second, and also Kodachrome "A" at one-fourth or one-tenth degree of a second. The image on the film is from 6.5 mm. and may be projected in slide form on a screen; the en-

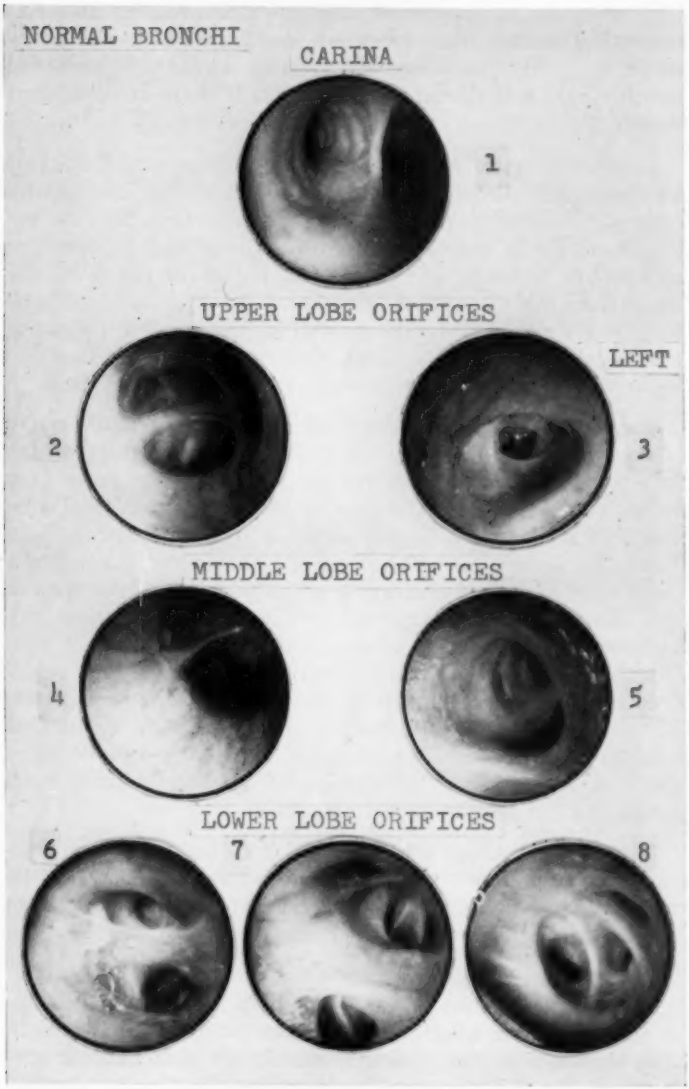
NORMAL BRONCHI
CARINA
1
UPPER LOBE ORIFICES
LEFT
2
3
MIDDLE LOBE ORIFICES
4
5
LOWER LOBE ORIFICES
6
7
8

Plate 7.

largement on paper or on film is from 24 mm. It requires the same time as does the development of cinematographic films in color, so that we make them only in exceptional cases; the cinematographic film being more economical and more interesting.

The photographs in black and white have the advantage of rapid development; they complete the filmed sequence. Ordinarily we have the print a few hours after the examination. Kodak + X films may be used, with an exposure time of one-twenty-fifth of a second. The grain is finer, and the exposure time remains sufficiently short. In all cases the image on the film has a diameter of 6.5 mm. We immediately make an enlargement on paper, which permits a perfectly clear 50 mm. image to be obtained. Enlargements as slides may also be made for projection.

The camera employed is a reflex type opening at 3.4 with a focus of 38 mm., and permitting a setting of 0.50 m. or 0.60 m.

The photographs shown here have not been retouched, intentionally; they represent those that the photographer (who has not specialized in medical photography) gives us. We be-

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Fig. 1. Tracheo-bronchial spur.

Fig. 2. Right upper lobe orifice viewed through the right angle telescope.

The apical branch is reduced to a slit visible at 3 o'clock. For better examination it is necessary to use a right angle telescope. The anterior orifice is visible at the top, the posterior in the middle of the field.

Fig. 3. A detail of the left upper lobe spur.

The lingular division is clearly visible through the right angle telescope; its dividing branches may be perceived at the bottom.

Fig. 4. Middle lobe orifice viewed through the direct telescope.

The middle lobe orifice is at 12 o'clock, a secretion is perceptible at the entrance. Below, the orifice of the inferior lobe bronchus.

Fig. 5. Orifice of the middle lobe viewed through the right angle telescope.

At 6 o'clock, the departure of the lower lobe bronchus may be detected.

Fig. 6. Bronchi of the right lower lobe viewed through the direct telescope.

At 9 o'clock, the medial basal is clearly evident.

At 5 o'clock, the superior orifice, with bubbling in evidence.

At 3 o'clock, the continuation of the lower lobe bronchus where spurs of division are perceptible.

Fig. 7. Right lower lobe bronchi.

At 5 o'clock, the superior orifice is very open. The first spur of the division is seen. At 11 o'clock, the departure of the medial basal.

At 2 o'clock, the termination in three branches: anterior basal, lateral basal, posterior basal.

Fig. 8. Same patient a little further: the "terminal bouquet."

From the center about 3 o'clock: the posterior basal; the lateral basal; the anterior basal.

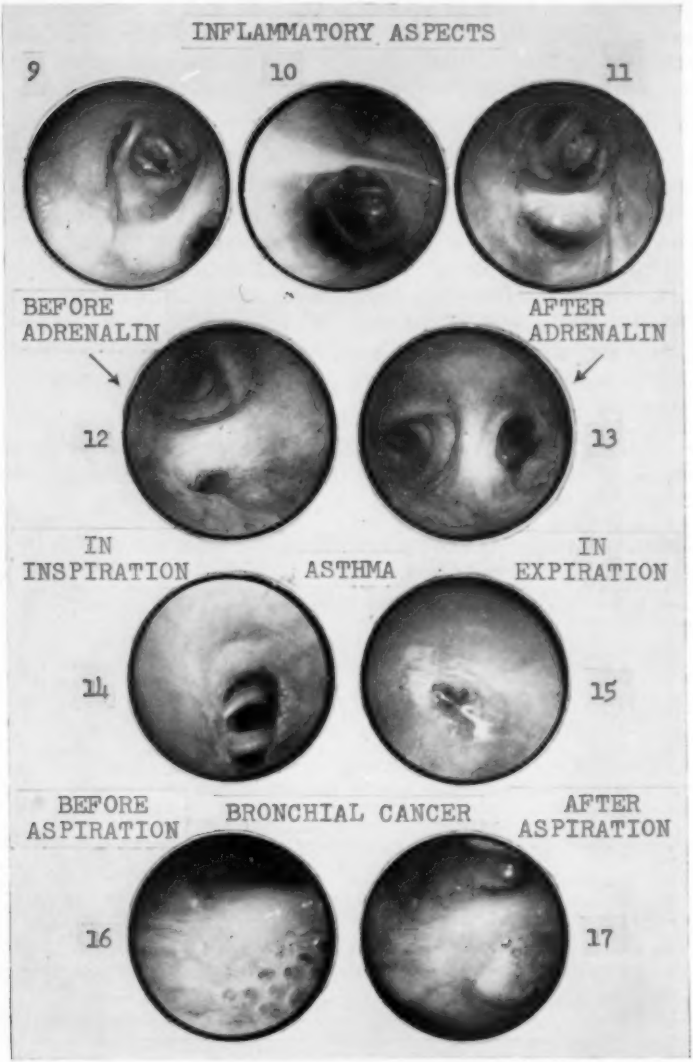
INFLAMMATORY ASPECTS
9
10
11
BEFORE ADRENALIN
AFTER ADRENALIN
12
13
IN INSPIRATION
ASTHMA
IN EXPIRATION
14
15
BEFORE ASPIRATION
BRONCHIAL CANCER
AFTER ASPIRATION
16
17

Plate 8.

lieve that the technique will be improved when a still more powerful illumination is achieved.

RECENT ACQUISITIONS AND PERSPECTIVES.

The same principle as that previously described for broncopy, rectoscopy, cystoscopy, laporoscopy, peritoneoscopy, choscopy is applied to the whole field of endoscopy: esophagosgastroscopy, can be performed with the same kind of apparatus. A modification of the apparatus was designed for the examination of organs which allow the use of a bigger tube, such as the rectum, the esophagus, and the stomach;[4] the lighting is double. Two light sources and two quartz rods are used. With a slight increase in the diameter of the tube, the lighting of the field of examination may be doubled (see Figs 6, 8).

With S. Segal we have published recently a special technique for esophagoscopy. We first introduce a cover tube made of transparent plastic. This is put into place in the patient, while he is sitting, with the help of a mandril; the technique is the

Fig. 9. Bronchi of the right lower lobe.

At 3 o'clock, the orifice of the superior branch; toward 12 o'clock, the medial basal; between the two, the terminal bouquet.

A certain inflammatory and edematous aspect is to be noted. The terminal branch is partially obliterated by an abnormal secretion (the left side is shown in Fig. 10).

Fig. 10. General view of the bronchi of the lower lobe.

At 11 o'clock, the left upper lobe bronchus orifice.

Toward 6 o'clock, the superior orifice; between the two, the left terminal bouquet, the classical axis of which is observed; symmetrical from the right, from outside to inside and from front to back. Inflammatory aspect with purulent secretions from the terminal branches. Bronchial dilatation.

Fig. 11. Detail of the left lower lobe of the same patient as in Fig. 10.

The superior orifice is open widely; the termination in three branches with purulent secretions from the lateral basal.

Fig. 12. Inflammatory stenosis of the left lower lobe bronchus.

At 10 o'clock, the upper orifice and the lingula very permeable and, at about 6 o'clock, the lower lobe bronchus orifice almost entirely stenosed.

Fig. 13. Same patient after local application of Adrenaline. Stenosis has partially regressed.

Figs. 14 and 15. These two figures are interesting because they clearly show the expiratory spasms in the course of asthma.

The first shows the aspect of the left lower lobe bronchus at the time of inspiration.

The second shows the same region in the course of expiration. The considerable reduction of the caliber may be seen at the elimination of bronchial hypersecretions.

Fig. 16. Aspect currently encountered in a patient with bronchial cancer.

At 12 o'clock, the right middle lobe bronchus orifice; at 4 o'clock, the right lower lobe bronchus orifice from which bloody bubbles are escaping.

Fig. 17. Same aspect after aspiration.

A tumoral formation of a whitish aspect is to be observed at the entrance of the right lower lobe bronchus; almost complete obliteration of this bronchus.

same as that used for introduction of a stomach tube. The patient is then put in the position of esophagoscopy. The mandril is removed and the esophagoscope can be introduced into the cover tube with security. This also avoids soiling the extremity of the tube as it passes through the mouth and throat. It is important that the cover tube be transparent, as it allows the vocal cords, the arytenoids, the opening of the esophagus and the mucus membranes of this region to be inspected as the instrument proceeds. This tube, shorter than the esophagoscope, does not impede the normal handling of the latter.

This procedure allows introduction of a rigid gastroscope into the stomach. This gastroscope can be used without difficulty for photography or cinematography.

ENDOSCOPY AND TELEVISION.

A recent experience has shown us the advantage of television.

Television can be used in the very room where the endoscopy is being performed, and a television camera can be adapted in the place of the eye or of the cinema camera. This allows everyone present in the room to follow the progress of the apparatus in the organ explored; moreover, the image, which the endoscopist and his assistants see, can be watched in the classroom of a medical school.

In this article we have explained the technique for bronchoscopy and esophagoscopy which we know very well. It is interesting to note that our technique of systematic endoscopic cinematography may be applied to all organs. In France we have specialists in rectoscopy and gastroscopy (S. Segal, M.D.), laparoscopy (Pergola, M.D.), colioscopy (Palmer, M.D.), cystoscopy (Jaupitre, M.D.), and others, and have founded an International Medical Society of Endoscopic Photography and Cinematography, with more than 300 members. The president is A. Soulas, M.D. (Paris); general secretary, J. M. Dubois de Montreynaud, M.D. (Reims). Dr. P. Holinger, of Chicago, and Dr Max Fourestier, of Paris, are honorary presidents.

SOME RECENT CINEMATOGRAPHIC FILMS.

1. New Technic of Bronchoscopy (systematic endobronchial cinematography), by J. M. Dubois de Montreynaud, R. J. Edwards and A. J. Gladu, Reims. There are four parts in this film: Bronchoscopic technique, bronchial tuberculosis, bronchial cancers and asthma (experimentation in animals and observations in humans), 277 mm.

2. Esophagoscopy (personal technique, with endoscopic motion pictures, by J. M. Dubois de Montreynaud, S. Segal and G. Fraisier).

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DIFFERENTIAL DIAGNOSIS OF HEARING PROBLEMS OF ADULT PATIENTS.*†

HARRIET L. HASKINS, A.M.,

Baltimore, Md.

In 1946 when Carhart analyzed the test findings of a group of patients at Deshon General Hospital, the speech hearing threshold and the average of the best thresholds in the band from 512 to 2048 c.p. showed a coefficient of correlation of about + 0.75. About one-third of all patients were found to have a difference in the two thresholds of 11 db or more. A survey of the patients over 14 years of age seen at the Hearing and Speech Center of Johns Hopkins Hospital during the past three years reveals that about 30 per cent have complex hearing problems for which routine tests give insufficient information. One suspects that there are many problems common to the subjects represented in the group Carhart statistically studied and those under audiologic evaluation at Johns Hopkins.

When routine audiologic test procedures fail to yield a consistent picture of hearing, it is necessary to inquire further into the nature of the patient's particular communication problem. The medical history, the otologic examination, the internal consistency of the battery of hearing tests and the observation of the patient's function in all communication must add up to a reasonable overall picture.

Time spent in obtaining a thoughtful, detailed medical history is often most rewarding. The patient's description of the sort of difficulty he has in communication may be very helpful. How he functions in a test situation may also be indicative of the nature of his problem. Does he arrive at threshold listening with ease and consistency? Is there an

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* Read at the Annual Meeting of the American Speech and Hearing Association held in Los Angeles, Calif., Nov. 19, 1955.

† From the Hearing and Speech Center, Department of Otolaryngology of the Johns Hopkins University School of Medicine and of the Johns Hopkins Hospital.

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indefinite range of loudness, often as much as 30 db, which might be considered threshold? Does he report that the levels where he admits hearing are really loud, and yet does not respond to slightly less intense levels? How does he function when speech sounds are comfortably loud for him? Is he confused by a moderate amount of noise when trying to understand speech? Does the patient respond to a variety of ambient noises of moderate loudness, and fail to understand speech unless he can see the speaker? Does he report that his hearing difficulty fluctuates markedly from day to day? All these behavioral factors make one question the reliability of the test results.

There are several other conditions which may indicate need for further study. The lack of response to all sound stimuli regardless of the intensity is legitimately very rare, for the incidence of total deafness is negligible. The person who has a mild to moderate amount of difficulty in communicating and has indicated threshold hearing at levels of 70-80 db has not given an accurate test picture. Patients who have extreme difficulty in understanding speech and yet repeatedly report that they hear the sound of the voice at quite low levels are in need of further study. It is found that their communication does not improve, either by the use of comfortably loud voice or amplification; a hearing aid apparently creates confusion of noise in the ear without promoting clarity. Then, too, the person who achieves a gain of 60-75 db with a hearing aid needs further investigation.

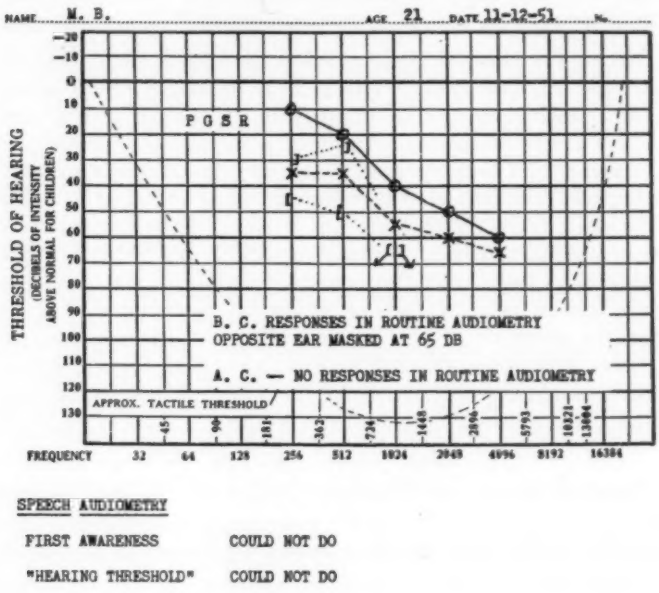
Three of the most useful measurements of hearing function are:

1. Threshold readings for a pure tone spread of at least 6 octaves.
2. The relationship between the responses to air conduction and bone conduction stimuli.
3. A test of ability to hear fine differences among speech sounds.

The third test, known as the test of discrimination, has proved the most useful of all. It is the best single index available for determining how effectively one can use his hearing. When the ability to discriminate fine differences among

speech sounds is markedly impaired, it is important to attempt to find the cause.

The use of psychogalvanic skin resistance audiometry provides pertinent information about the function of the end-organ of hearing. This test by no means gives all of the facts which one should like in some complex cases, but it does give one more measurement that is free from various behavioral complications. Such additional information often makes possible a more accurate prognosis for the patient and aids in planning an appropriate course of therapy.



SPEECH AUDIOMETRY

FIRST AWARENESS	COULD NOT DO
"HEARING THRESHOLD"	COULD NOT DO

Fig. 1.

It is worthwhile to consider the records of a group of patients for whom the results of a test by psychogalvanic skin resistance audiometry provide a critical measurement of hearing function and help to clarify the interpretation of the overall picture. Some of the problems found among the group considered here are those with a history of meningitis, lues, psychological disorders, familial hearing impairment, condi-

tions that appear to be the result of vascular sclerotic changes, long-term severe impairment and auditory perceptive and language disorders.

The first (Fig. 1) is a 21-year-old girl who suffered a severe, undiagnosed illness at the age of 12. A form of encephalitis or meningitis was suspected. She had a fever of 104° for several days and was unconscious for periods during this time. On the tenth day of this illness, tinnitus developed, and she lost her hearing. She was able to finish high school with honors. On examination, one notes marked

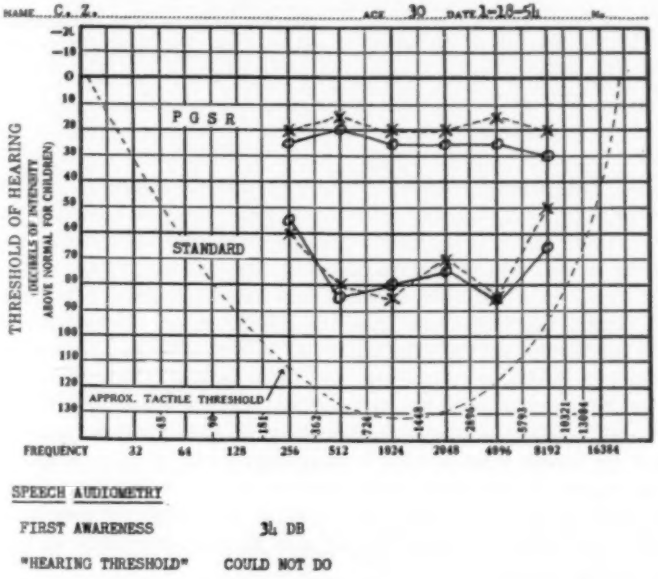


Fig. 2.

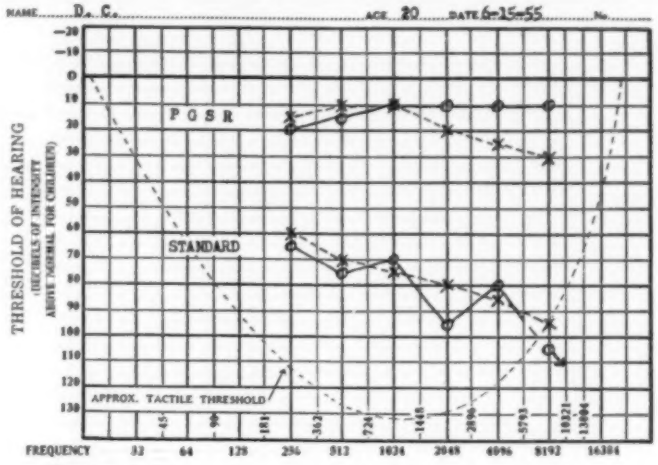
changes in her voice quality. She appears to be an unusually good lipreader. She reports that she does not hear any sounds regardless of the intensity level. There were no subjective responses to pure tone stimuli by air conduction. There was a subjective response to bone conduction stimuli at 512 cp during the initial test only. The measurement obtained by psychogalvanic skin resistance audiometry indicates a moder-

ately severe bilateral impairment of the end organ of hearing; however, one could not be sure that this problem was not further complicated by a disorder of auditory perception. Because of this question and other manifestations of behavioral disorders, the patient was referred to a psychiatrist. He observed that, under hypnosis, she could understand a conversational voice of moderate intensity by audition alone. It is clear that this problem involves both a moderate organic impairment and a functional disorder.

Fig. 2 shows the audiometric findings for a 30-year-old man who had been treated periodically for 13 years for lues. There were no other pertinent medical findings. He reports that he has had difficulty hearing for about eight years. He has tinnitus and reports that at times his hearing fluctuates. He attempts to use a hearing aid sporadically but cannot understand speech by auditory cues with or without the aid. Sometimes mild amplification creates only noise in his ear. The patient's speech is good, and his language understanding by visual cues and language usage are also good. Note the levels of response reported by routine audiometric procedures. These responses were inconsistent, and it was difficult to determine a threshold measurement. On three different occasions the patient indicated an awareness of the sound of the voice at about 30-34 db. This is a less complicated listening situation for the patient than those of pure tone and speech hearing audiometry. The test by psychogalvanic skin resistance audiometry reveals that the patient has no worse than a mild impairment in the function of the end organ of hearing. His problem is severely complicated by difficulty in auditory perception. Hearing aid use is of minimal benefit to this patient and he must continue to rely on visual cues.

Fig. 3 is a 20-year-old boy who was first examined while attending a school for the deaf. He is reported to have congenital deafness. His mother and her brother have like hearing problems. This boy responds to a great variety of ambient noises but does not understand speech by auditory cues. His own speech is limited, but it is articulated within a normal time span. He enjoys using a mild gain hearing aid. His language understanding by lipreading and his speech have

improved noticeably since he left school and has spent most of his time with hearing people, and has had lessons in speech and language work. Note the first awareness response level of 16 db above normal and the audiogram by psychogalvanic skin resistance of near normal level. This does not appear to be a problem of impairment of the end-organ of hearing, but one of auditory perception and/or language association.



SPEECH AUDIOMETRY

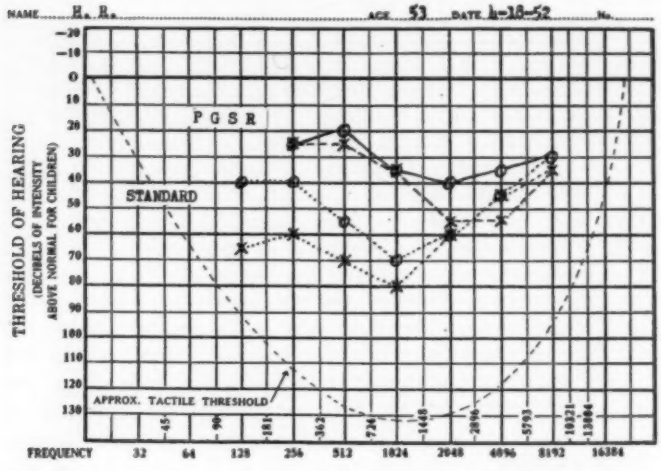
FIRST AWARENESS 16 DB

"HEARING THRESHOLD" COULD NOT DO

Fig. 3.

The fourth case (Fig. 4), is a 53-year-old woman who is a personnel counselor in a large high school. When first seen she was in poor physical condition and worried about her rapidly progressive hearing impairment, which she feared would soon incapacitate her for her position. She has a positive family history of "progressive deafness" and had anticipated serious problems herself. Her father was deaf for many years and was a very poor communicator. This patient was attempting to use a powerful hearing aid when we first saw her. The noise in the instrument and the amplified sound about her were very distracting. She had ceased to mingle

socially and often stayed away from work because it was an exhausting experience. Note the awareness level of 26 db which is consistent with the audiogram made by psychogalvanic skin resistance method; also, the great variance in her discrimination scores. This patient has a moderate bilateral nerve type hearing impairment. After medical treatment her general condition was improved. She was assured of the relatively moderate degree of her impairment and

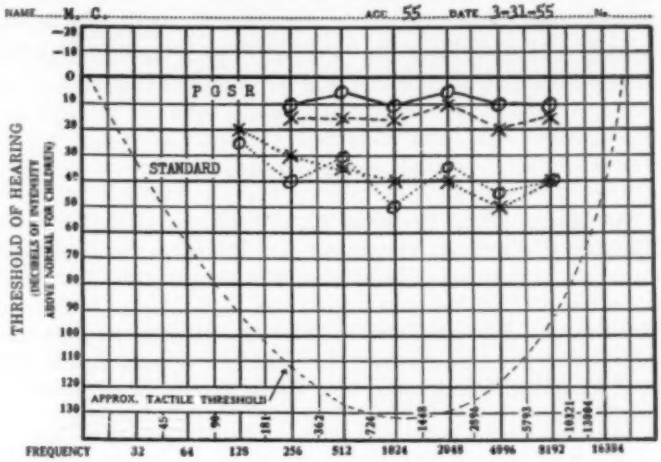


SPEECH AUDIOMETRY		DISCRIMINATION TESTS *	% LOSS
FIRST AWARENESS	26 DB	AT SLOW RATE IN QUIET	56
"HEARING THRESHOLD"	50–62 DB	AT NORMAL RATE IN QUIET	26–68
		AT NORMAL RATE IN NOISE	—

Fig. 4.

advised that any progressive aspect of her hearing loss could not be determined. She was fitted with an instrument appropriate to her degree of loss and counselled in its use. A more recent test shows a speech hearing threshold of 44 db instead of the original 50-62 db, and with her new aid she achieves a threshold of 14 db. and a mild loss of 12 per cent in discrimination. This is reasonably good function for this degree of end-organ impairment.

Fig. 5 refers to a 55-year-old woman who has had difficulty hearing for three years. The otologic examination is negative. There is no history of ear infection nor of a familial hearing impairment. She does have tinnitus. She communicates fairly well in an easy listening situation, when the background noise is minimal and one person nearby is speaking to her in a moderately loud voice and at an even rate. Attempts to use a hearing aid have only complicated her problem. Note the



SPEECH AUDIOMETRY		DISCRIMINATION TESTS *	% LOSS
FIRST AWARENESS	26 DB	AT SLOW RATE IN QUIET	14
"HEARING THRESHOLD"	46 DB	AT NORMAL RATE IN QUIET	28
		AT NORMAL RATE IN NOISE	56

Fig. 5.

near normal thresholds by psychogalvanic skin resistance audiometry; also, that awareness for sound is 20 db. better than the speech hearing threshold. A difference of 6 to 10 db is usually observed in subjects whose audiometric test is relatively flat. Note the marked loss in discrimination of 28 per cent when test items were presented at normal rate in quiet. This seems to be a case of phonemic regression. The end-organ of hearing is functioning within range of normal

limits. It appears that in this case the difficulty in understanding speech is probably caused by changes in the auditory perceptive mechanism.

The use of psychogalvanic skin resistance audiometry is a special and exacting technique used in the diagnostic process at a medical center. It appears to test the function of the end-organ of hearing. It provides a measurement which assists in differentiating between the receptive hearing function and the combined function of receptive and perceptive hearing mechanisms. This information is invaluable to the otologist and audiologist in understanding the patient's problem and in planning a feasible course of therapy.

ANNOUNCEMENT.

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956. Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

THE FUNCTION OF A MEDICAL SOCIETY SPONSORED HEARING CENTER.*

HAYES A. NEWBY, Ph.D. (By invitation),

Palo Alto, Calif.

We specialists in the field of audiology feel a very close and warm relationship with you who practice the medical specialty of ear, nose and throat. We like to feel that we are all working together in the dedication of our efforts for the betterment of individuals with impaired hearing. I have been asked to speak on the topic of "The Function of a Medical Society Sponsored Hearing Center." Before getting into this specific topic, however, I feel it would be worthwhile to spend some time reviewing the development of this new specialty of audiology and the growth of the community hearing and speech center.

Fifteen years ago there were no hearing and speech centers, and the word "audiology" was unheard of. The first "hearing and speech centers" were the military aural rehabilitation centers of World War II days, where, for the first time, the combined abilities of various specialists were concentrated on the diagnosis and treatment of hearing impairment. In these military centers the meaning of the words "diagnosis" and "treatment" was extended far beyond their usual medical connotation.

In the extension of the meaning of "diagnosis" and "treatment" lies the definition of clinical audiology. Audiology is concerned with "diagnosis" from the standpoint of measuring the hearing function of an individual and predicting the success of surgical procedures or prostheses designed to overcome his hearing loss. Audiology is concerned with "treatment" in

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* Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, San Francisco, Calif., January 21, 1956.

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the sense of providing rehabilitation in the form of auditory training, speechreading, and speech training for those who have permanent impairment of their hearing function.

Much has been written and said about the lineage of this new field of audiology. While it is true that its ancestors include various medical specialties, psychology, physics and education, audiology is a direct descendant of the fields of speech correction and otology. The other fields I mentioned may be "kissing cousins" of audiology, but its "mammy" and "pappy" are speech correction and otology. Sometimes audiology dwells with the one parent and sometimes with the other. For example, at the University of Pittsburgh audiology is a division of the Department of Otolaryngology in the medical school, while at Northwestern University audiology is part of the Department of Speech. Sometimes audiology leaves the parental home to live alone, though with close ties both to mother and father; for example, the Portland Center for Hearing and Speech, the Seattle Hearing and Speech Center, and the San Francisco Hearing and Speech Center. It is with these community audiology centers, rather than the university sponsored centers, that this paper is primarily concerned.

To carry the analogy further, audiology is the product of a "marriage of convenience." During the war the medical departments of the Army and Navy asked for assistance in setting up programs of aural rehabilitation. For this assistance medicine turned to university speech clinics and recruited specialists in speech correction, experimental phonetics, and in the education of the deaf. Together with otologists these non-medical specialists devised tests for measuring the social efficiency of hard-of-hearing individuals; tests for malingering and other types of functional deafness; tests for the selection of hearing aids; and they planned a program of rehabilitation for those who were handicapped because of their hearing impairment. The success of the combined efforts of the medical and non-medical specialists was measured in the excellent record established by the armed forces in returning hard-of-hearing and deafened service men to duty, or returning them to civilian life with a minimum of handicap. Neither group of specialists could have accomplished this job single-handedly; together they created the field of audiology.

You are all familiar with the post-war development of audiology and the mushrooming of audiology clinics, or hearing centers, which today have become hearing and speech—or speech and hearing—centers. The techniques of assessment of hearing function, selection of hearing aids, and rehabilitation of the hearing-handicapped which were developed during the war have been improved and extended in these civilian centers. Perhaps the outstanding post-war innovation in audiology has been the focusing of attention on the training of pre-school deaf and hard-of-hearing children and their parents. In most centers, children make up a considerable proportion of the case load.

During the war, audiology programs were under the direction of medical officers according to the military table of organization. When the war was over the otologists who had been concerned with aural rehabilitation programs returned to their civilian practice. Many of these otologists, being well aware of the accomplishments of the military audiology program, desired to see a similar program established for the civilian population. Thus most hearing and speech centers today owe their existence to the pioneering efforts of one or more otologists.

The administrative direction of community audiological programs has largely passed from the otologist's hands, however. This is a natural development, since there are few otologists whose sole interest is in audiology or who can afford to devote the major share of their efforts to the administration of audiology programs. During the war it was different; then otologists were ordered to duty, and they had no private practice. Post-war audiology programs were usually started on a financial shoestring and depended on public support for their continued existence. The direction of such an audiology program required more time than most otologists could give; so, while otologists were largely responsible for the creation of community audiology centers, they were forced by circumstance to relinquish administrative control to non-medical specialists, and since the war the development of community audiology centers has been primarily along non-medical lines.

Just as the interest of otologists in the field of audiology was stimulated by their participation in the military aural rehabilitation programs, so also was the interest of the non-medi-

cal specialists who, together with the otologists, had pioneered in the military centers. These non-medical specialists, most of whom had been recruited from college speech clinics, realized that audiology offered them new opportunities for service for which their previous training and experience had to some extent prepared them. Some of these non-medical specialists returned to their universities to organize and direct training programs in audiology, and some became the administrative directors of community hearing and speech centers. What before the war had been university speech clinics, shortly after the war became speech and *hearing* clinics. The impact of the growth of audiology along non-medical lines is nowhere evidenced more forcefully than in the reorganization in 1947 of the American Speech Correction Association into the American Speech and Hearing Association, and the change in the title of that organization's official publication from the *Journal of Speech Disorders* to the *Journal of Speech and Hearing Disorders*. By 1950, extensive graduate training programs leading to the M.A. and Ph.D. degrees in audiology, or in speech and hearing, were in existence in a dozen of the leading American universities. The products of these training programs were ready, willing and able to assume clinical and administrative responsibilities in community hearing and speech centers. The field of audiology had "come of age."

Most otologists have accepted in good grace this development of audiology as a non-medical specialty and have been content to assume an advisory capacity to audiology centers in medical matters, welcoming the extension of audiological services to the general public which the centers have made possible. There are some otologists, however, who have played the role of the over-protective parent who hates to see his child grow up and assume independence. Such otologists deprecate the contributions of the centers which they helped to found and look down their noses at the non-medical clinicians who perform the day-to-day work at these centers. Some feel that in helping to develop the specialty of audiology they have created a Frankenstein monster which is now completely out of control. Fortunately, these deprecators and complainers are in the minority; the great majority of otologists are playing the role of well-adjusted and proud parents who have accepted audiology as a trusted and valued professional partner.

In all fairness to the complainers it must be admitted that some audiology programs have developed improperly and unprofessionally. In the creation of a new field it was inevitable that there would be mistakes. Some community centers were brought into existence by overly-ambitious individuals who were more interested in expansion for expansion's sake than in sound professional growth. In some centers there was far too little medical supervision; but by and large, hearing and speech centers around the country have developed soundly and with excellent medical guidance.

So much by way of introduction to the specific title of my paper. I want now to tell you how one community audiology center was created and how it has developed. I believe that the San Francisco Hearing and Speech Center, of which I am Director, has the unique distinction of being the only audiology center founded by a medical society. In 1948 the San Francisco Center was created through the joint efforts of the San Francisco Medical Society and the San Francisco Hearing Society. Professional guidance of the Center was provided by a committee of six otologists appointed by the President of the Medical Society. Originally, the Director of the Center was responsible solely to the committee of otologists. A little less than a year ago the Center was chartered as a non-profit corporation. The administrative control is now vested in a board of directors on which otologists are represented. The President of the Medical Society continues to appoint a Hearing and Speech Center Committee, which is now composed of four otologists and a pediatrician. This committee serves in an advisory capacity to the board on medical matters pertaining to the Center.

The function of the San Francisco Hearing and Speech Center is to provide specialized audiological services for referring physicians and medical agencies. One of the services offered by the Center is diagnostic audiometry, which means the assessment of the hearing function as an aid to the otological diagnosis of a hearing impairment. Diagnostic audiometry now includes also the examination of very young children who are suspected of having hearing impairment because of their failure to develop oral language. A second service is assessment of the hearing function in individuals who are candidates for the fenestration or the stapes mobilization opera-

tions, and the evaluation of the results obtained through these surgical techniques. A third service is hearing aid consultation—the advising of patients primarily as to whether they would be benefited by using any hearing aid, and secondly, making recommendations of a more specific nature in regard to hearing aids. In the fourth place, the Center provides rehabilitative services on an individual or group basis for children and adults with permanently impaired hearing. Finally, the Center operates a training program for pre-school deaf youngsters and their parents.

At present the Center is serving a case load of approximately 1200 patients a year. No patient is accepted without a medical referral, and 60 per cent of our case load comes on referral from private physicians. In the course of a year referrals will be received from as many as 125 different physicians in the Bay Area. Twenty-five per cent of the case load is referred from various governmental agencies, such as the Bureau of Crippled Children Services of the State Department of Health, the Bureau of Vocational Rehabilitation, and the Welfare Department. The remaining 15 per cent of patients are referred through other medical agencies, primarily ear, nose and throat clinics in various hospitals in the Bay Area. In every case, reports of the Center's examinations and recommendations are made to the referring physician or agency, so that there is close liaison between the Center and the medical referring source.

In 1948 the Center's staff consisted of one audiologist who performed his own secretarial work. From this modest beginning, the Center has developed a staff of eleven people, consisting of a Director, an Associate Director, a Research Director who doubles as a clinical audiologist, four other clinical audiologists, a test assistant, a nursery school teacher who works also part-time as a bookkeeper; an office manager, and a stenographer. At the San Francisco Center there is no resident otologist. In addition to the physicians on the medical advisory committee who are available for consultation, there is a pediatrician who is appointed by the President of the Pediatrics Section of the Medical Society to serve as medical advisor to the pre-school training program.

Like most activities of its kind, the San Francisco Hearing

and Speech Center has been beset by financial problems from the time of its founding. Rehabilitation is an expensive business and can never be self-supporting if services are to be available to all who need them. The San Francisco Center obtains only one-third of its support from patient fees. The rest of its budget comes from civic-minded organizations and individuals who have appreciated the value of the Center's contribution to the hearing-handicapped. Prominent among its supporters have been the Junior League of San Francisco, the Rotary Club of San Francisco, Infant Shelter, the San Francisco Hearing Society, and Mount Zion Hospital, where the Center is presently housed. Financial support has also been received from various foundations and governmental agencies. Most hearing and speech centers receive Community Chest support. To date, the San Francisco Center has not been affiliated with the Chest, although it is contemplated that application for Chest membership may soon be made.

The description of the San Francisco Hearing and Speech Center's activities would not be complete without paying tribute to the otologists who have been instrumental in its founding and development. I am pleased at this opportunity to acknowledge our debt of gratitude and continuing appreciation to Drs. Robert C. McNaught, Robert C. Martin, Walter P. Work, Shirley H. Baron, and William T. Duggan.

In many respects the history and development of the San Francisco Hearing and Speech Center parallel the history and development of hearing and speech centers in all parts of our country. The growth of the audiology center as a community responsibility and activity stems from the interest of otologists whose visions of a civilian aural rehabilitation program originated from their experience in the military service. In addition, of course, the development of the community center is due to the interest and vision of non-medical personnel whose basic training for the most part was obtained in the field of speech correction. Thus audiology is the child of parents of somewhat different background. I feel that both parents can look with pride on the accomplishments of their precocious child, and I believe that the future of audiology lies with its continuing allegiance to both parents. I hope that audiology will always obey the Biblical injunction to "Honor thy father and thy mother."

REVIEW OF THE CORTICOSTEROIDS—THEIR APPLICATION TO OTOLARYNGOLOGY.*†

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Hydrocortisone is the most important steroid secreted by the adrenal cortex. Like cortisone and the newer synthetic compounds (prednisone and prednisolone), it is a so-called gluco-corticoid and has only slight mineral and water retaining activity. In the remarks to follow, the terms corticoids, corticosteroids, and gluco-corticoids will be used interchangeably to designate hydrocortisone or any of the cortisone-like hormones.

Approximately 100 mm. of hydrocortisone are secreted in 24 hours by the intact, normal, human adult. The main function of this hormone is the preservation of healthy tissue and healthy cells, not only in normal physiologic conditions, but also during stress of any kind. Stressed tissues send impulses to the hypothalamus, which in turn stimulates the anterior pituitary to secrete ACTH. The latter stimulates the adrenal cortex to secrete hydrocortisone. Increased secretion of ACTH also results when the blood level of hydrocortisone is low. A third stimulus to increase the secretion of corticotropin from the anterior pituitary is the direct action of adrenalin, the hormone from the adrenal medulla.

In this function of maintaining homeostasis, hydrocortisone may produce many physiologic actions. I have reviewed these actions in a recent paper[1] and therefore, will not repeat them now. Instead, an attempt will be made to elaborate on only three actions of the corticosteroids which seem especially important in the field of otolaryngology. These three actions are: *1.* anti-inflammatory action; *2.* action in regard to infection, and *3.* action on capillary and connective tissue permeability.

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Before reviewing these three subjects, a short resume on the anatomy and physiology of the connective tissue seems in order. Loose connective tissue, such as that found under the epithelium of the nose, sinuses and most of the respiratory tract, consists of a homogenous gel-like matrix in which are inmeshed a mass of fibrils and fibers, cells, lymphatics and capillaries. We must not forget that the capillary bed is a part of the connective tissue. Another important fact, which was recently emphasized by Dougherty,[2] is that no capillary or blood vessel supplies nourishment directly to any cell in the body. Passage through the ground substance of the connective tissue is essential before the cells of any organ are reached by anything from the circulation; hence, the circulation in the ground substance is almost as important as that in the vascular bed. The most important cellular elements of the connective tissue are the fibroblasts and mast cells.

The mast cells are large granular basophilic staining mesenchymal cells, which, according to Asboe-Hansen,[3] function in forming the ground substance. The granules of these cells contain histamine, and also a heparin-like mucopolysaccharide which is a hyaluronidase inhibitor. Stress of any kind causes the mast cells to "spit" their granules into the gel of the connective tissue, thus liberating these substances. The corticosteroids also degranulate these cells. The mast cells have recently taken on great importance in regard to connective tissue, and undoubtedly are essential in maintaining the integrity of the ground substance. The connective tissue under the epithelium, lining the nasal membranes, contains many mast cells. The fibroblasts are more concerned in the forming of the fibrous elements of the connective tissue. The ground substance is normally a gel and is composed of protein and carbohydrate molecules. These molecules are polymerized (aggregated) to form mucopolysaccharides.

The most important of the polysaccharides is hyaluronic acid. The entire connective tissue is metabolically active and, hence, is in a continual state of flux. In other words, it is being constantly built up and constantly broken down. Enzyme action accounts for most of this activity. For example, the ground substance is built up by enzymes from the mast cells and, possibly the fibroblasts, and is broken down, liquefied or

depolymerized by the enzyme hyaluronidase. Thus, hyaluronidase changes the ground substance from a normal gel to a more or less of a sol. While hyaluronidase, in any important amount, has not been found in ground substance, Mayer[4] has shown that in inflammation there is a forty fold increase in this enzyme; hence, its precursor must be present in the connective tissue at all times. The consistency of the ground substance is of great importance to our physiology and our normal defenses. As stated previously, nourishment, carbon dioxide and oxygen, as well as metabolites and substances secreted by parenchymatous organs, must go through the ground substance before reaching their destination; also, invading bacteria and viruses, after passing through the epithelium, must go through the ground substance before reaching the blood stream or before reaching deeper important structures. For example, a virus which gets through the nasopharyngeal epithelium, must penetrate the ground substance before getting into the vascular bed; therefore, if the gel is firm, it provides a good barrier against invasion of these infecting organisms. A more liquid edematous ground substance allows a better chance for invasion. Some of the important streptococci, pneumococci and staphylococci produce hyaluronidase themselves and, hence, are particularly invasive. Other points about the connective tissue will be pursued further in the observations to follow.

THE ANTI-INFLAMMATORY ACTION OF THE CORTICOSTEROIDS.

One of the most important, interesting and incompletely understood actions of the glucocorticoids is their anti-inflammatory action. Due to this action, Selye[5] has named this group of corticosteroids *antiphlogistic hormones*. This is in contradistinction to the phlogistic hormones such as thyrotropin and growth hormone.

Dougherty[2] has brought out the fact that inflammation always occurs in the connective tissue. This very important fact has not been generally recognized. When one peers into an inflamed nose, whether the inflammation is of allergic, infectious or traumatic nature, the swelling is not in the epithelium, but is in the edematous ground substance of the connective tissue under the epithelium. In inflammation, we get an

increase in the connective tissue cellular elements, along with an increase in leukocytes and other wandering cells; also, the ground substance becomes edematous, and the capillaries engorged. The corticosteroids depress all of these processes.

Now, what is inflammation? It is a chain reaction[5] in the connective tissue, which is the result of stress. The stress produces cell damage which liberates certain substances which set off and continue the inflammatory reaction. The damaged cells are mainly fibroblasts and mast cells. Not all of the released substances are known, but among them are histamine, hyaluronidase, heparin-like mucopolysaccharides and leucotaxin. Thus, local inflammation and generalized stress differ only in degree and localization of the stress.

Dougherty[6] gives the following hypotheses as possible mechanisms in corticoid anti-inflammatory action:

1. Decrease in the level of circulating fibrinogen, thus producing a decrease in the amount of fibrin at the site of inflammation.

2. Inhibition of hyaluronidase, thus decreasing the action of the spreading factor.

3. Inhibition of the formation of hyaluronic acid of the ground substance.

4. Shedding of mast cell granules with release of a heparin-like mucopolysaccharide, which has anti-hyaluronidase action and which also binds histamine.

To this list, could be added the action of maintaining a good capillary bed. Dougherty[6] believes that all of these actions take place, but that there may be a more fundamental anti-inflammatory phenomenon involving the action of hormones on the many substances liberated from cell damage.

INFECTION AND CORTICOSTEROIDS.

There is no question but that the gluco-corticoids in *large* doses impair the host's resistance to many infections. There are numerous explanations for this. Thomas[7] states that there are at least six possibilities. They are as follows:

1. A Direct Action on the Infecting Organism. There is no direct evidence that this takes place.

2. An Effect on the Neutrophils. Although the corticosteroids produce a systemic leukocytosis, fewer pus cells migrate to the area of local inflammation. There is slight evidence, but no definite proof, that the bactericidal properties of leukocytes are diminished. Some virus infections are also enhanced by *large* doses of corticoids and since available evidence shows that neutrophils are not important in virus infections, the leukocyte hypothesis becomes somewhat untenable.

3. Influence in Antibody Formation. There is no definite evidence that this is a factor in humans.

4. Interference With the Inflammatory Reaction. The corticoid depression of tissue inflammation is an accepted fact, but as noted above, the explanation is uncertain. At any rate, the anti-inflammatory theory does not explain the enhancement of virus infections, nor the enhancement of bacteria which are injected directly into the blood stream. In these two instances, there is no local inflammatory reaction.

5. The effect on serological mechanisms, such as the nonspecific hyaluronidase inhibitor.

6. Damage to the Reticuloendothelial System. The macrophages in this system are for the purpose of clearing the blood stream of bacteria and particulate matter. There is some evidence that like certain colloids, the corticoids block the cells of the reticuloendothelial system so that these macrophages then cannot clear the blood stream of infectious organisms.

It can readily be seen that the phenomenon whereby *large* doses of corticoids enhance infection, is not easily explained by any one hypothesis.

On the other side of the picture, Robinson,[8,9] et al., Vollmer[10] and Lurie,[11] et al., give evidence to show that in *small* doses, these hormones not only fail to enhance infection, but also aid the infected host and protect the host from infection. Robinson states that to date many patients have received cortisone, and the number of reports citing intercurrent infection has been negligible.

Jawetz[12] states that in patients seriously ill with infection and toxemia, the antitoxic effect of cortisone may save the

patient while a proper antibiotic is destroying the infection.

It would appear that, as far as infection is concerned, with doses of corticoids large enough to produce symptoms of hyperadrenocorticoidism, there is enhancement of the infection; and that with very small doses, or with short term dosage, there may be improvement of the host with inhibition of the infection.

I have used this fact, probably advantageously, by giving small doses of hydrocortisone or ACTH for a short time in the early stages of virus upper respiratory infections. The combination of ascorbic acid and one of the bioflavonoids, also firms the connective tissue gel, by antihyaluronidase action,[13] and hence, can be used with corticoids in this connection.

CORTICOSTEROIDS IN RELATION TO CAPILLARY PERMEABILITY AND CONNECTIVE TISSUE PERMEABILITY.

The work of Zweifach[14,15,16] has shown that the exchange of substances between the blood stream and parenchymatous tissues or epithelial structures is a much more complicated matter than had previously been described. In order better to visualize it, some of the anatomy and physiology of the peripheral vascular bed will be described.

In the first place, the capillary endothelium and the inter-endothelial cement substance are not the only structures involved in this matter. There are at least five structures involved, and they are grouped together under the term hemato-parenchymal barrier. They are as follows:

1. A Thin, Slimy Proteinaceous Substance Lining the Inside of the Capillaries. This apparently comes from the blood plasma. Zweifach's experiments show that if it is washed away, some edema will occur.

2. The Endothelial Cells. While water and large molecules pass through the inter-endothelial cement, carbon dioxide, oxygen and lipid soluble substances diffuse mainly through the endothelial cells; hence, the maintenance of healthy endothelial cells, which the corticoids aid in doing, is very important in providing nourishment and oxygen to the cells of the important structures in our specialty.

3. Inter-endothelial Cement. This substance is probably a calcium proteinase and is secreted continuously by the endothelial cells. Endothelial cement makes up only a very small amount of the total capillary surface (about one per cent), but is important in the passage of water and large molecules. Proteolytic enzymes will break down the cement substance, but hyaluronidase will not. The pH of blood markedly affects the permeability of this structure, and, adequate calcium is necessary in order to maintain its normal consistency; however, Zweifach has found that hypoxia does not increase its permeability. It was formerly thought that practically all substances passed through the cement substance, but as noted previously, oxygen, carbon dioxide and lipid soluble substances pass mostly through the endothelial cells.

4. Peri-capillary Sheath. This structure is of the utmost importance, particularly in relation to edema. It is a condensation of fibrils and very firm ground substance which supports the capillary. When it is firm, it prevents edema. When it is liquefied by hyaluronidase, edema occurs. The corticoids help to keep this structure firm and prevent edema. Ascorbic acid and the bioflavonoids also aid in this regard.

5. The Connective Tissue. Like the peri-capillary sheath, the consistency of the connective tissue ground substance is also affected by the corticoids.

It can be seen that in the production of edema, several of these structures may be involved; also, all of these structures are in a constant state of metabolic flux and vary considerably in their physical and chemical make-up. At times they are more permeable than others. Of special importance is the peri-capillary sheath. This structure exerts the ultimate control of the capillary permeability and the latter's permeability characteristics are regulated largely by the corticosteroids.

There is another factor about edema which has nothing to do with capillary permeability. This factor is that of the action of hyaluronidase on the ground substance. Since hyaluronidase is liberated in inflammations, it liquefies the ground substance and, thus, produces edema directly. This is probably an important factor. Since corticosteroids are anti-hyaluronidase factors, their action helps prevent edema in this

manner; thus, the production of edema, whether it be from allergy, infection or trauma, is a complicated matter and much more complex than simple osmosis and hydrostatic pressure.

The contraindications to the use of the corticoids and ACTH are too well known to dwell on them at this time; however, I emphasize the fact that large doses of these hormones over long periods of time will depress the adrenal cortex. To me, this is the most dangerous factor in their use; therefore, in otolaryngologic practice, whenever possible, we should not resort to this type of therapy. In surgical cases, however, if there is any possibility of cortical depression from previous therapy, prophylactic corticoids should be given before and for a few days following surgical stress; also, intravenous hydrocortisone should be available for emergencies.

OTOLARYNGOLOGIC CONDITIONS IN WHICH THE CORTICOSTEROIDS AND ACTH ARE USEFUL.

1. Hay Fever.

Particularly in severe cases not controlled by ordinary methods and in localities where the season is short. Besides systemic therapy the local use of hydrocortisone eye drops is effective. The local use of hydrocortisone intranasally is only mildly effective.

2. Status Asthmaticus.

In this serious condition, intravenous hydrocortisone or intravenous corticotropin may be a life saving procedure.

3. Penicillin and Other Drug Reactions, Hives and Angioneurotic Edema.

These maladies are usually self limited and respond well to the corticosteroids.

4. Acute Edema of the Larynx.

Whether the edema is from infection or trauma, the use of the corticoids may prevent the necessity of doing a tracheotomy.

5. Critically Ill and Toxic Patients.

The corticosteroids can tide the patient over while antibiotics and other supporting measures are carried out.

6. Idiopathic Midline Granuloma of the Facial Tissues.

ACTH and the corticosteroids are the most effective treatment in this serious condition.

7. Erythema Multiforme.

In many such cases, lesions in the mouth are the first sign of this disease; hence, the otolaryngologist may be the first to see the patient or may be called in consultation. Corticosteroids with appropriate antibiotic therapy give relief and shorten the course of the disease.

8. Pemphigus.

Like erythema multiforme, the first signs of pemphigus are often lesions in the mouth. Until the era of corticosteroids and corticotropin, this disease has been considered to have a fatal outcome. The author has been consulted in three cases of pemphigus. All were treated with corticotropin, corticosteroids and antibiotics. Two are now well on no therapy and one is free of symptoms on a maintenance dose of hydrocortisone.

9. Histamine Cephalalgia.

If histamine and other vasodilating types of therapy fail to give relief, corticosteroids, and particularly intravenous corticotropin, will often bring about improvement; however, vasodilating therapy should be continued.

10. Contact Ulcer of the Larynx.

Cauterization or fulguration of the lesion followed by a short course of corticosteroid therapy is well justified in this condition. Granulation tissue is diminished, and epithelium can more readily cover the ulcer.

11. Laryngeal Polyps and Extensive Nasal Polyps.

The following regime is recommended in these conditions:

a. Surgical removal of the polyps.

b. A short course of post-operative corticosteroid therapy.

c. Elimination of etiological factors.

d. Appropriate allergic treatment.

12. Fresh Stricture of the Esophagus.

Less scarring results if the corticosteroid therapy is initiated immediately. Standard treatment should also be followed.

13. Eczema and Dermatitis of the External Ear.

Local use of hydrocortisone with the appropriate antibiotic is very effective in these cases.

14. Bell's Palsy.

In early cases of Bell's Palsy, we have had very good results with the use of vasodilators combined with corticosteroids and a low-salt diet.

15. Meniere's Disease.

We are now treating Meniere's Disease in a manner similar to that of Bell's Palsy. That is, a combination of vasodilatation, corticosteroids and low-salt diet. The author's experience is not large enough to comment accurately upon the effectiveness of this form of treatment in Meniere's Disease.

16. Pre-operatively and Post-operatively in Patients With Depressed Adrenal Cortices. (This has been commented upon.)

As a matter of fact, in many operative cases, it is probably worthwhile to give a few doses of corticosteroids before surgery and for a day following surgical stress.

17. Local Spray and Mist Hydrocortisone Therapy in Respiratory Allergy.

We have had only moderate success with this local type of therapy.

18. Post-surgical Edema.

In surgical cases where post-operative edema is often a problem, corticosteroids can be effectively used. The author has found these hormones especially useful following nasal reconstructive surgery.

19. Status Lymphaticus.

This condition is supposedly due to a hypo-activity of the adrenal cortices and may result in death from very mild stress. Death from anesthesia furnishes the most typical example. If

intravenous hydrocortisone could be administered early enough, recovery could be expected.

20. Anaphylactic and Other Forms of Shock.

Intravenous hydrocortisone is one of the most effective measures.

21. Emergency Surgery in Debilitated Patients.

When it becomes necessary to do surgery in debilitated patients, stress hormones should be administered just before and a few days following surgery.

22. In Patients With a Poor Stress Mechanism.

In a paper recently given at the meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago, I stated that it seemed to me that there were many patients with a poor stress mechanism, who gave normal findings on all of the tests now available for this function. In other words, there were no present tests sensitive enough to determine this mild hypofunction. I mentioned that these patients could not well tolerate stress of any kind. For example, they would develop a stuffy nose and sore throat when exposed to drafts, fog, emotional stimuli and so forth. They were subject to headaches and other vasomotor symptoms. Since that time, one of our colleagues called my attention to an article by Tintera[17] on "The Hypoadrenal State and Its Management". Tintera had observed 200 patients in the above category, and he stated that the regular tests, such as the eosinophil test and checking on the corticoid metabolites in the urine, were always negative. He found that the sugar tolerance test was often abnormal. After eating rapidly absorbed carbohydrates, their blood sugar level would rise to hyperglycemic levels, but this would be followed by prolonged hypoglycemic levels. It was during the latter state their symptoms were more marked. He believed that stresses stimulated their overactive parasympathetic system to stimulate the pancreas to secrete excessive insulin. This kept them in a hypoglycemic state. He recommended the following treatment:

1. Where possible, elimination of stresses,
2. Bellergal to block the parasympathetic system,
3. Whole adrenal extract, and
4. Antihypoglycemic diet.

Tintera's article has increased my confidence in this idea, and we are now treating some of these patients in the above manner with certain modifications. Instead of whole adrenal extract, we use an occasional injection of ACTH. Desensitization is undertaken where indicated in the allergic patients. Ascorbic acid with bioflavonoids is used in conjunction with the above therapy.

Further experience and more observations will be necessary before expressing a definite opinion on this group of patients.

If the corticosteroids and ACTH are used in otolaryngologic conditions in doses producing no permanent harm to the tissues and functions of the body, we will find that we now have some very useful tools which were not at our disposal a few years ago.

SUMMARY.

Hydrocortisone is the most important hormone secreted by the adrenal cortex, and like other gluco-corticoids, has the function of maintaining homeostasis.

Since knowledge of the anatomy and physiology of the connective tissue and peripheral vascular bed is necessary in order to understand the corticosteroid actions, a brief review has been given. The action on inflammation and infection has been discussed. The relation of the gluco-corticoids to capillary and connective tissue permeability has also been commented upon. Great emphasis has been placed upon the depression of the adrenal cortex by large and prolonged dosage of corticoid therapy. Otolaryngologic conditions in which the corticosteroids and ACTH are useful have been listed. The possibility of a certain group of patients in the hypoadrenal state, unable to be diagnosed by present laboratory methods, has been discussed.

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ELONGATED STYLOID PROCESS EVALUATION OF SYMPTOMS AND TREATMENT.*†

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Case reports of symptomatic elongation of the styloid process have long appeared in the literature, receiving consideration as a medical rarity. In more recent years investigators have attributed a widened spectrum of symptoms to styloid elongation and find that with increased attention the diagnosis is not infrequently made in patients previously labeled as psychoneurotic. This paper is presented in the cause of wider recognition of symptomatic styloid elongation, and to add possibly new symptoms to the syndrome.

HISTORY.

Although ossified stylohyoid ligaments had been reported by anatomists for centuries, Weinlecher, of Vienna, is credited with the first surgical removal of a symptomatically elongated styloid in 1872. Stirling[13] in America in 1896 described three cases, one his wife, later successfully operated; and sporadic case reports continued to appear. In 1932 Thigpen[14] reported 11 cases, with some treated by surgical resection and some by fracturing the elongated styloid outwards by digital pressure through the throat. Clinical results with styloid fracturing were not favorable. Morrison[11] in 1940 stated that styloid fracturing "may or may not influence the neuralgia," and Lederer[9] in 1942 noted that "fracture of the styloid process during operations on the tonsils usually produces annoying symptoms." Reports on styloid resection, however, were good, and Turner[15] in 1936 stated empirically that styloid removal would be followed by cure.

In 1937 Eagle[4] reported excellent results from styloid resection in two cases, but Shambaugh in editorial comment on his

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paper, in the Eye, Ear, Nose and Throat Year Book, sounded a word of caution that neck infections had followed previous cases with reported spread of infection to the middle ear and meninges. Since the antibiotic era, however, no mention of complications has been noted in the literature reviewed.

Since 1940 many new symptoms have been attributed to elongated styloids. Fritz[7] in 1940, and Eagle[5,6] in 1948 and 1949 reported over 250 cases of the Duke University group, many of which had intraoral resections with excellent results. Eagle noted about 12 cases that, as well as local throat symptoms, demonstrated pain of external or internal carotid artery distribution. At surgery actual impingement of these vessels could be demonstrated in some cases, with relief of the symptoms on styloid resection. Eagle attributed this to actual arterial compression, or to pressure on sympathetic nerves in its wall. In 1951 Schmidt[12] added a case of throat tightness, hoarseness, restricted voice range and break, and vocal fatigue in a professional singer cured by resection of bilaterally elongated styloids. Douglas[2] in 1952 reported a case of severe third division trigeminal pain that increased on swallowing, and was relieved by resection of an elongated styloid.

Most writers have done intraoral resections mostly with Crowe-Davis gag exposure under general anesthesia, although Johnson[8] used a combination of sodium pentothal and local anesthetic. Loeser and Cardwell[10] reported one case in 1942 operated by an external incision similar to Mosher's, behind and below the angle of the jaw, where the elongated styloid was more laterally placed producing carotid symptoms, and could not be palpated easily in the tonsillar area.

ANATOMY.

Detailed anatomical studies of styloid ossification and anomalies by Dwight[3] in 1907 described the development of the styloid in the second branchial bar from the cartilage of Reichert, with a proximal ossification center, the tympanohyal, and a distal stylohyal center. The proximal center, according to Cunningham,[1] appears shortly before birth and fuses with the petro-mastoid during the first year. The stylohyal center appears shortly after birth and ossifies slowly, fusing with the upper portion after puberty, or fusion may never occur. In

some cases it may become continuous with the inferior cornu of the hyoid bone. Anatomists give normal styloid length as 2.5 cm., and state that abnormally elongated styloids are present in about 4 per cent of persons, occurring bilaterally in about half of these. Only a small portion of these elongated styloids are thought to be clinically symptomatic, however.

A styloid of normal length passes between the external and internal carotid arteries, so that abnormal internal or external deviation may cause pressure on either vessel, even

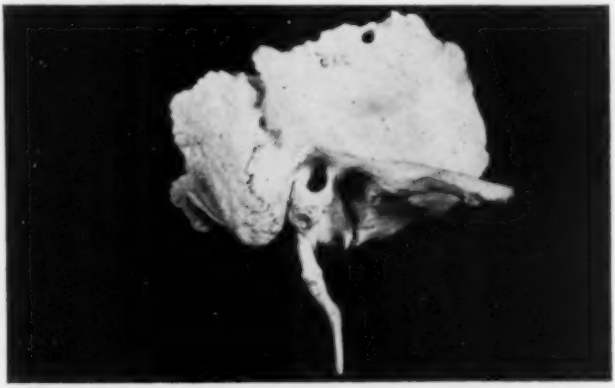

Fig. 1. Specimen showing configuration of elongated styloid. (From collection of Washington University, St. Louis.)

though the process is not elongated. The tractions applied to the styloid by the attachments of the styloglossus, stylopharyngeus, and stylohyoideus muscles and the stylohyoid and stylomandibular ligaments probably play an important role in directing the deviation in growth of the elongated styloid tip. The frequently occurring ram's-horn configuration shown in an anatomical specimen in Fig. 1, correlates with X-ray studies, in the writer's cases, that the proximal anterior deviation may result from traction of the stylomandibular ligament and styloglossus muscle, and the distal downward angulation may result from the pull of the stylohyoid ligament and muscle.

The anatomical specimen in Fig. 2 demonstrates the rela-

tionship of the facial nerve and stylomastoid foramen to the styloid process. It was vividly impressed on me during post-graduate training never to succumb to the natural impulse to twist and break off an elongated styloid tip with a hemostat, if one is found presenting into the tonsil fossa at surgery. Cases were cited where such a maneuver resulted in backward and lateral fracture of the styloid near the base similar to the breaking of a wishbone, and complete severance of the facial

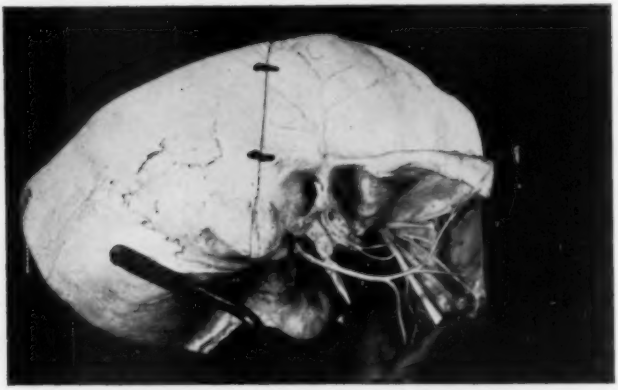

Fig. 2. Specimen showing relationship of facial nerve to the styloid process. (From collection of Washington University, St. Louis.)

nerve. Although I cannot find an actual report of such an incident in the literature, the mechanics of such an accident, especially with the oblique line of fusion at the base of the styloid shown in Figs. 1 and 2 substantiate this hazard. The writer has taken care to remove styloid tips with minimal twisting or pressure to the proximal portion.

SYMPTOMS.

The most typical history of elongated styloid pain is sticking or soreness in the throat following tonsillectomy with the reaction by the patient that the throat had not healed, though many cases occur long after tonsillectomy, or in unoperated throats. The pain is usually steady, dull-aching and nagging, frequently with increased shooting pain in the throat or to the

ear on swallowing. At times a sensation of a foreign body sticking in the throat is noted, or there may be a sensation of drawing or soreness of the throat muscles. Symptoms may follow areas of distribution of disturbed fibers of the Vth, VIIth, IXth or Xth cranial nerves.

The pain attributed by Eagle to internal carotid pressure localizes especially in the parietal and orbital areas, and external carotid pain localizes in the temporal area and the lower side of the head and below the eye.

DIAGNOSIS.

The diagnosis is suggested by the history and is usually made by digital palpation of the firm bony elongated styloid tip in the tonsillar area. As a styloid of normal length does not reach this area, any palpable styloid is abnormally elongated although not necessarily symptomatic. Many elongated styloids are diagnosed on routine examination, or by X-ray, in patients with no symptoms, and their removal is certainly not indicated. In general it has been noted that anteriorly and medially deviated styloids are more likely to be symptomatic, and that more laterally placed elongations, especially those connecting with the hyoid or with a calcified stylohyoid ligament, tend to be asymptomatic. In several of the writer's cases styloids were palpable bilaterally, but were only unilaterally symptomatic on the side of the slightly longer and medially displaced tip.

Consistently in my cases the patient has shown a marked reaction to digital pressure on a symptomatic styloid tip, with such remarks as "that is it" or "that's the spot," and relate that the maneuver caused an exacerbation of pain identical to that of his complaints. X-rays are routinely taken before surgery but were found necessary for diagnosis in only one case where a large tonsil tag was present and the elongated styloid more laterally placed and causing carotid symptoms. In this patient lateral pressure through the tonsil tissue reproduced the symptoms, and though there was a palpable sensation of deep firm resistance the tip could not be clearly outlined.

Granger position anterior-posterior views give the best comparative view of their lengths and their medial or lateral devia-

tion. A lateral view centered behind the angle of the mandible at "soft-tissue" exposure, with the patient's mouth closed and jaw thrust forward so as to flex the neck but extend the head at the atlanto-occipital area gives the best anatomical detail and evaluation of the actual length. Standard lateral X-rays taken for cervical spine, sinus, or skull series may fail to demonstrate large styloids. Fig. 3 shows such a film read as nega-

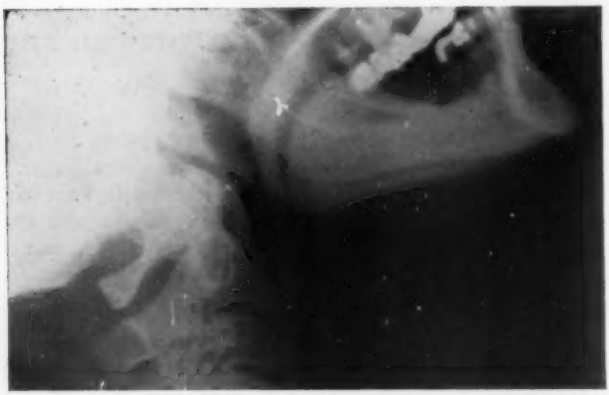

Fig. 3. Conventional lateral view of cervical area.

tive for styloid elongation by an excellent radiologist, but a repeated film, taken in the above-mentioned position (see Fig. 4), clearly shows a markedly elongated styloid.

Differential diagnostic problems most frequently encountered are: Costen syndrome with mandibular joint disturbances, cervical spine arthritis with radicular pain, atypical migraine, impacted third molars or other dental disease, tonsillar or pharyngeal abscesses or neoplasms, sphenopalatine ganglion neuralgia, and glossopharyngeal, trigeminal or vagal neuralgias.

REPORT OF CASES.

Since 1950 the writer has done seven trans-oral styloid resections on six patients. One patient had bilaterally elongated styloids with symptoms only on the right side, which a right

styloid resection relieved in 1951. Fourteen months later she developed almost identical symptoms on the left side and returned for successful resection of her left styloid. One patient who had mandibular joint tenderness had increasing Costen syndrome symptoms worse with chewing, in the two years since her surgery, though the local styloid tenderness and soreness in the throat were relieved. Four of the patients had had

Fig. 4. Special lateral view for demonstrating styloid.

complete tonsillectomies, and two had large tonsil tags. During this same period at least this number of additional patients were seen with palpable elongated styloids who were not operated upon because of absent, mild, or not definitely diagnostic symptoms.

A 32-year-old woman recently seen in consultation with her neurosurgeon, Charles Spicer, M.D., presented symptoms so interesting as to warrant mention at this time, even though no definite conclusion can be drawn unless planned styloid resection clears her symptoms. Her now-pertinent history is that in November, 1955, she had increasing shooting pains about the left side of her head, followed by sensations of "hot water running over the left side of her head" and a slight sensation of numbness in this area. At this time she had slight drooping of the left eyelid of the pseudoptosis type, and constriction

of the left pupil. No definite enophthalmos or unilateral sweating was noted, and complete neurological examination was otherwise noncontributory. X-rays showed markedly elongated styloids of the pseudojointed type bilaterally which were laterally placed. The lateral neck pain was reproduced by deep pressure through her unoperated tonsils, and on deep palpation in a tender area behind the angle of the mandible.

Clinically, this strongly suggests pericarotid sympathetic plexus interference by an elongated styloid as the basis for her Horner's syndrome and pain of carotid artery distribution. This would emphasize sympathetic plexus involvement more than arterial compression in the production of her symptoms. She is the only case I have seen in which external surgical approach may be more advisable than transoral.

SURGICAL TECHNIQUE.

All patients so far have been operated by transoral approach with general endotrachial anesthesia and exposure with the Crowe-Davis mouth-gag and position. In the two patients with tonsil tags the styloids could be exposed easily through a vertical incision behind the tonsil tags, and tonsil tag removal that was not otherwise indicated was not done. Vertical incision was made through the mucosa over the presenting tip, and the styloid exposed by careful blunt dissection with a hemostat and finger palpation. The muscular and tendonous attachments were found most easily and safely separated with a fairly dull flat hook elevator made by bending back the tip of a Freer submucous elevator. When the tip is freed as high as possible, working under intermittent exposure and finger palpation, the distal end is firmly clamped with a tonsil hemostat, to prevent losing it in the pharyngomaxillary space, and the tip resected as high as possible with minimal twisting or side pressure with a long gooseneck mastoid, or universal handle sphenoid rongeur. Usually a 1.5 to 3.5 cm. portion is removed. No bleeding has been encountered, and the incision is closed with about two small plain catgut sutures.

Patients are given wide-spectrum antibiotic coverage for about four days after surgery. One patient with hay fever and sneezing spells developed barely palpable emphysema crepitus in the lateral neck following surgery, but no definite

neck infections have developed. As a precaution patients are warned not to blow their noses for several days after surgery, to reduce the chance of cervical emphysema or neck infection. The operative site heals with little edema of the lateral throat, and the patients experience less discomfort than from a tonsillectomy.

SUMMARY.

A clinical evaluation of elongated styloid process symptoms and treatment has been presented, with consideration of historical background, anatomy, diagnosis and treatment. The possibility that Horner's syndrome may result from styloid pressure on cervical sympathetic fibers of the carotid plexus is suggested.

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Professional Building.

CONGENITAL HEMANGIOMA OF THE LARYNX.*†

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and

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Hemangiomas of the skin of the face and neck are common tumors in the infant, but are not so common in the larynx. As recently as 1944, Ferguson[1] made a review of the literature and found but seven cases. To these he added one of his own. Dargeon and Daly[2] in 1951, reviewed the literature and found ten cases of hemangioma of the larynx. They reported a case, including the autopsy findings. Since 1951, five infants were seen and treated for congenital hemangioma of the larynx at Babies Hospital. All of these patients had lesions elsewhere on the skin of the face or neck, in addition to the laryngeal hemangioma. They were admitted to the otolaryngological service because of obstructive laryngeal dyspnea. The pathology, symptomatology, diagnosis, and treatment of these cases will be presented.

PATHOLOGY.

Boyd[3] classifies hemangiomas into two main types: capillary hemangioma and cavernous hemangioma. Capillary hemangioma includes port wine stains, birthmarks and strawberry marks. Cavernous hemangioma includes the group made up of structures similar to erectile tissue. They contain large and irregular spaces and lack the definiteness of fully formed blood vessels.

Hertzler[4] has classified hemangioma into three groups: capillary, venous and cavernous. The capillary hemangiomas are intradermal capillary varicosities which may invade the deeper structures. The venous hemangiomas are elevated tumors

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which are present at birth or appear soon after. They may develop rapidly during the first few months of life. The cavernous hemangiomas show large spaces that lack the form of mature blood vessels.[5]

In our group all of the patients had hemangiomas about the face, neck or oral cavity, in addition to a laryngeal lesion. The youngest patient was one month and the oldest four months of age. There were four females and one male, which is different from the cases reported in the past. The literature indicates that males outnumber the females two to one.

Sweetser[6] stated that infantile hemangiomas of the larynx were usually subglottic in location, and in adults the lesions were more apt to be on the vocal cords or above this level. In our series, supraglottic lesions were found as frequently as those in the subglottic area or trachea.

McMahon[7] reviewed Sturge-Weber disease. He reported a case with laryngeal disease. In this disease there are extensive hemangiomatous nevi of the skin and cerebral hemangioma.

There was one pathological report in our group of five patients, and this was a capillary hemangioma. The patient died, and tissue removed at autopsy from the trachea was reported as follows:

"On cross section, the tissue is dark red in color and homogenous. It is hemangiomatous tissue. This forms a layer between epithelium and cartilage and is symmetrical. The maximum thickness is 3 mm. where the diameter of the trachea is 7 mm. and one has the impression that when completely filled with blood it probably occluded the lumen completely. The histologic appearance of the lesion in the trachea resembles more nearly the so-called hemangioendothelioma of the infantile variety, where the lesion appears more solidly cellular with no or relatively few dilated vascular channels."

Hemangiomas vary in size at different times depending upon the blood content of the tumor. This was evident in the repeated direct laryngoscopies on these patients. At times the hemangioma was large, and on other examinations it was scarcely visible.

SYMPTOMATOLOGY.

Suehs and Herbut[8] listed the laryngeal symptoms in patients that had previously been reported. Obstructive laryngeal

dyspnea was the outstanding finding. In our group, although the patients had lesions on the lip, tongue and oral cavity, the symptoms of stridor and obstruction of laryngeal origin dominated the clinical picture.

Laryngeal symptoms were not present at birth. As the hemangiomas of the face, lip, tongue or oral cavity became more prominent and appeared to grow, the laryngeal symptoms became more evident. There was noisy breathing, obstructive dyspnea and disturbances in swallowing. The degree of obstruction was variable. At times it was very slight, only to become severe at another period. This was probably due to the changing blood volume of the hemangioma. The patients failed to gain weight. It was necessary to do a tracheotomy in all five patients.

DIAGNOSIS.

The patients were referred to the otolaryngological service because of laryngeal symptoms. The presence of hemangioma in the larynx was determined by direct laryngoscopy. In this group of five infants, two were subglottic and tracheal in location, and the remaining three involved the epiglottis, aryepiglottic folds and the mucosa over the arytenoid cartilages.

The appearance of the hemangioma by direct laryngoscopy varied, depending upon the blood content of the tumor. In each instance the lesions were large enough to produce laryngeal obstruction. There were no biopsies taken, due to the danger of hemorrhage. A pathologic report was obtained on the patient who died. The lesion was classified as a capillary hemangioendothelioma.

TREATMENT.

Hendrick[9] discusses the therapy of vascular tumors. He includes solid carbon dioxide snow, irradiation, electrosurgery, injection of sclerosing solution and tattooing or neutralization of color. The method of therapy depends upon the age of the patient, type and location of the lesion and its rate of growth.

Kasabach and Donlan[10] reported two cases treated successfully by Roentgen therapy.

TABLE I.

Case	Location of Tumor	Hemangioma Elsewhere	Method of Diagnosis	Treatment	End Result
E.W.	Supraglottic	Face and Lip	Laryngoscopy	Tracheotomy Radiotherapy	Decannulated
L.B.	Subglottic, Tracheal	Mediastinum	X-ray Bronchoscopy	Tracheotomy Radiotherapy	Good but mediastinal hemangioma still enlarging
C.C.	Supraglottic, Base of tongue, Epiglottis and aryepiplottic folds, trachea	Skin and structures of jaw, neck and pharynx	Laryngoscopy	Tracheotomy Radiotherapy	Died suddenly Hemangio-endothelioma
J.S.	Supraglottic Subglottic	Neck, ear and soft palate	Laryngoscopy Bronchoscopy	Tracheotomy Radiotherapy	Decannulated
A.V.	Supraglottic	Lip, tongue, pharynx, Eustachian tubes, middle ear, aryepiglottic folds and arytenoids	Laryngoscopy	Tracheotomy Radium Radiotherapy	Good

TABLE II.

Case	Sex	Age in Months	Outstanding Symptoms	Duration
E.W.	F	4	dyspnea	7 days
L.B.	M	2½	dyspnea	8 days
C.C.	F	1	dyspnea	1 day
J.S.	F	1	dyspnea	2 days
A.V.	F	1¼	dyspnea	2 weeks

X-ray therapy was selected for the laryngeal lesions in our group. This type of treatment is not without danger to the infant larynx. It is important to keep the dose low and to spread it out over a period of several months. Each patient received an average of 400 Roentgens within a period of nine days. This was repeated at intervals of three months until a total of 1200 units had been given. There was no evidence of any X-ray complication to the larynx as a result of this therapy. The lesion appeared to become smaller after completion of the treatment.

The response to X-ray treatment is difficult to evaluate. Capillary hemangiomas have a tendency to disappear spontaneously after a few years. It is possible that patients might do just as well if the laryngeal lesions are left alone. An exception would be the lesion that is growing rapidly. One of our group has involvement of the mediastinum as well as the larynx and trachea. It has not been possible to control the mediastinal lesion with X-ray therapy.

All of the patients needed a tracheotomy. These patients required a long stay in the hospital and a great deal of nursing care. In order to cut down the expense involved, the parents were taught how to change the tracheotomy tube and to take care of the patient at home.

RESULTS.

Four of the five patients are living. One died of respiratory obstruction, and at autopsy the lesion was found to involve a large portion of the trachea. Two patients have been decannulated. One will be decannulated in the near future.

The remaining case has extensive involvement of the mediastinum in addition to the larynx and trachea. The prognosis in this patient is questionable.

The lesions that are present on the face will require further therapy which will be supervised by the plastic surgeon.

SUMMARY.

Congenital hemangioma of the larynx is probably more common than is indicated by reports in the literature.

Tracheotomy is necessary if laryngeal obstruction is present.

X-ray therapy may be of value in these lesions. Precautions are necessary when it is used.

The prognosis is good if the lesion is not too extensive and if a vital structure is not involved.

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A RARE CASE OF CHRONIC OTORRHEA WITH INTRACRANIAL COMPLICATIONS.*

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C. M., a 73-year-old man, came to the office because of a running ear. Within three months he had developed intracranial complications that caused his death. The diagnosis was not made until the autopsy findings were completed. Later it was learned that there are laboratory tests whereby an accurate diagnosis might have been made earlier.

A brief review of this patient's clinical course will bring out some of the diagnostic problems encountered:

The patient was a retired teamster who had had an intermittent discharging right ear since childhood. Another otologist reported that the ear was dry one year before, but that it had been discharging six weeks before the patient first consulted me on October 6. As he had a large amount of granulation tissue in the middle ear, some of this tissue was removed for microscopic examination. This tissue was reported as being a granuloma. X-rays of the mastoid made at this time showed them to be sclerotic, with no definite bone destruction. He was treated at the office for the next three weeks, but as he had an increasing amount of pain and headache, he was hospitalized on October 28.

His internist had treated him for some time for diabetes, which was controlled with insulin. He also had a mild hypertension. Medical and neurological examinations were otherwise essentially negative. A lumbar puncture and spinal fluid were normal. X-rays of the mastoid on October 28 showed no change from those made on October 6. He was given 8,000,000 units of Penicillin within the next six days.

A radical mastoid operation was done on October 30. The

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soft tissues over the mastoid and ear were very vascular and somewhat indurated. The mastoid cavity was extremely small, and there was very little pus present. Granulation tissue in the mastoid antrum and middle ear was removed for microscopic examination. This tissue again revealed a granuloma. The dura was exposed and found to be normal. There was no evidence of extension over the lateral sinus area. There was no VIIth nerve paralysis immediately following surgery.

His postoperative course was very stormy. He had chills and fever, and his headache persisted. X-rays of the chest revealed a pneumonia, which responded to treatment within a week. He began to develop weakness of the VIIth nerve and had difficulty in swallowing; he also had some hoarseness. There was a paralysis of the right side of the palate as well as the right vocal cord. A lumbar puncture and spinal fluid were again negative. At times he was fairly alert mentally, but at other time he was stuporous.

On November 7 a neurosurgical consultation reported that there was no evidence of increased intracranial pressure, in spite of the fact that the VIIth to the XIIth nerves on the right side were involved. The neurosurgeon stated that such lesions are most often due to malignancies extending along the base of the skull and originating in the nasopharynx or immediate vicinity. He found no evidence of a posterior fossa abscess or mass. He noted that there was slight tender swelling at the tip of the mastoid, and he suggested that later a biopsy might be taken from a gland in this area.

On November 9 the blood culture was reported negative. The sensitivity tests of cultures made from the ear showed the organisms to be most sensitive to Chloromycetin. As previously stated, the patient had received 8,000,000 units of penicillin during the first six days of hospitalization. The penicillin was stopped and 250 mg. of aureomycin was given every four hours for one week. When the ear culture showed the organisms to be sensitive to chloromycetin, he was given 250 mg. of this drug every six hours for the next ten days; and Gantrisin for two weeks; however, none of these drugs produced a definite improvement in the patient.

His general condition gradually became worse. His tem-

perature was irregular, although not very high. He had so much difficulty swallowing that it was necessary to feed him through a Levine tube. He had a constant headache. At times he was lethargic or stuporous. X-rays of the skull on November 18 were reported as showing no evidence of involvement of the petrous tip and no evidence of destruction at the base of the skull. These X-rays did suggest a mass in the nasopharynx. (See Fig. 1.)

Because of the patient's poor mental and physical condition it was difficult to examine the nasopharynx well, and a definite tumor mass had not been seen. The pathologist who performed the autopsy six weeks later reported that no mass could be palpated in the nasopharynx. On November 20, with strong retraction of the soft palate, a small definite mass about one cm. in diameter was seen high in the vault on the right side of the nasopharynx. The surface of the epithelium seemed intact. A large amount of this rather soft friable tissue was removed for microscopic examination. This tissue was reported as a granuloma, similar to the three specimens previously removed from the ear canal, the mastoid antrum and the middle ear. Several pathologists examined these specimens, and all agreed on the diagnosis. On December 18 X-rays of the skull were repeated; the report stated that no change had occurred in the past month, and that there was no evidence of involvement of the petrous tip or foramina at the base of the skull. (See Fig. 2.)

The patient's condition rapidly became worse, and he expired on January 6, three months following my first examination. Fortunately, an autopsy was done. The following is a brief resume of the autopsy findings relating to this case:

"A mass about 3 cm. in diameter and 1 cm. thick, with very ill-defined borders, surrounded the entire right petrous tip area and extended into the foramen magnum. This mass enveloped the cranial nerves of the right side. It had dissected downward near the mid-line to a point just above the nasopharynx on the right but apparently had not penetrated through the lining of the nasopharynx on this side. As previously noted, palpation of the nasopharynx by the autopsy surgeon revealed no abnormality. The bony petrous area was mushy in consistency and had the same type of material as

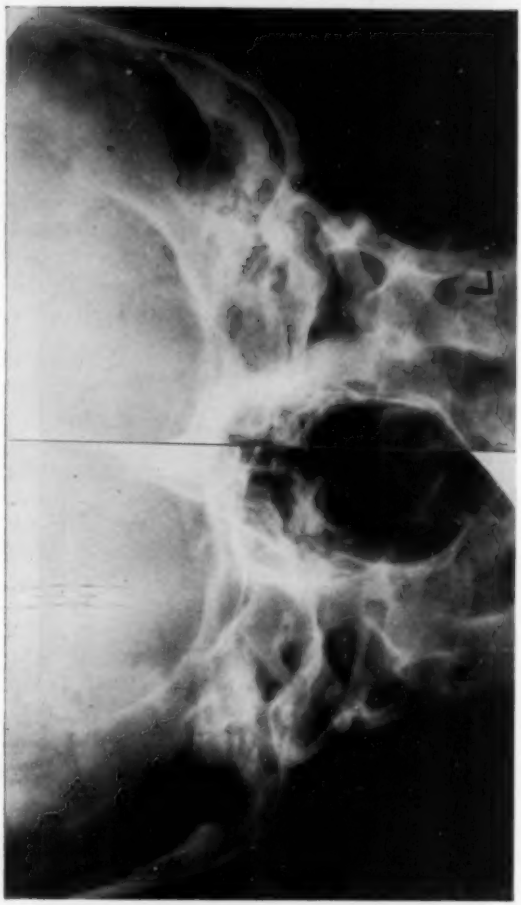

Fig. 1. X-ray of the petrous ridge on December 18th, 3 weeks before patient's death, showed no evidence of bone destruction, although the autopsy showed almost complete destruction of this area.

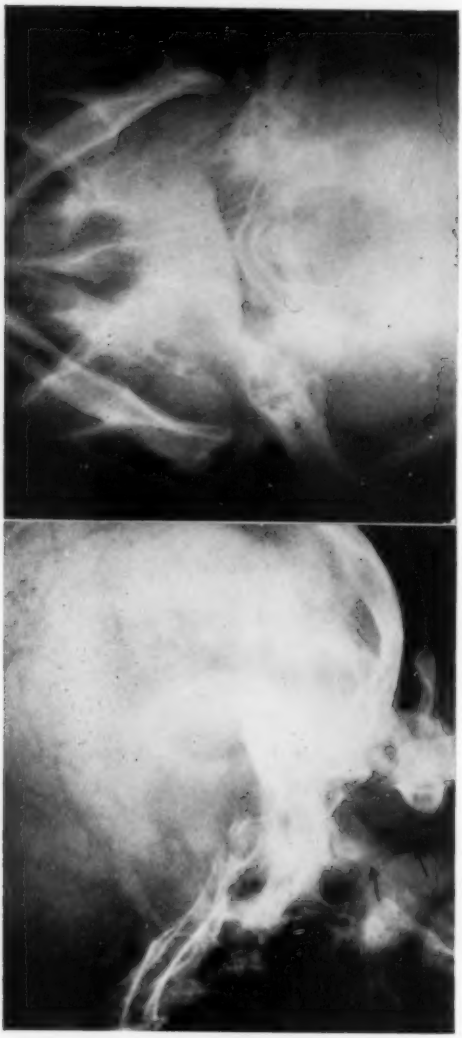

Fig. 2. X-rays of the skull on December 18th, 3 weeks before patient's death, showed no definite evidence of involvement of the foramina of the base of the skull. A mass in the nasopharynx was "suggested" by the Roentgenologist, but this is not seen clearly in the X-rays.

the mass, which was a thick, rubbery, reddish-yellow mass, resembling granulation tissue. There was also a thrombus of the right lateral venous sinus. The gross impression was that of a neoplasm, but when the microscopic sections of this mass were examined, they revealed many areas with very typical actinomycosis." (See Fig. 3.)

Actinomycosis of the ear is very rare. Risch[1] in 1938 reviewed the literature and found only 31 cases involving the ear, and only 12 of these cases involved the middle ear. Nowack[2] reported two cases in 1938. Townrow[3] reported a case in 1945 that was remarkably similar in all aspects to my case. Schubert[4] reported one case in 1951, and Bistrenin[5] reported a case.

The fact that actinomycosis of the ear is so rare is of academic interest, but it is of much more interest to our specialty that actinomycosis, as well as other diseases that produce granulomatous lesions, are rather commonly found in the mouth, nose, throat and neck. The fact that a differential diagnosis can be made between actinomycosis and these other diseases, by making bacteriological studies of this granulomatous tissue, makes it imperative today that we are familiar with these tests in order to determine which specific drug or antibiotic is to be used.

Months later I learned that laboratory tests might have enabled us to make this differential diagnosis earlier. While attending an instruction course given by Thomas B. Fitzpatrick, M.D., Professor of Dermatology at the University of Oregon Medical School, we were told that whenever one had a chronic granuloma which had not been identified, this tissue should be ground up and cultured for a fungus, etc. He was referring to an article written by Lyle A. Weed, M.D., and David C. Dahlin, M.D.,[6] of the Bacteriology and Surgical Pathology Division of the Mayo Clinic.

Dr. Weed and Dr. Dahlin reported 14 cases illustrating some of the common errors made in trying to establish an etiological diagnosis of tissue removed when only a histological examination was made. Their work was based on the generally known facts that certain organisms elicit a fairly characteris-

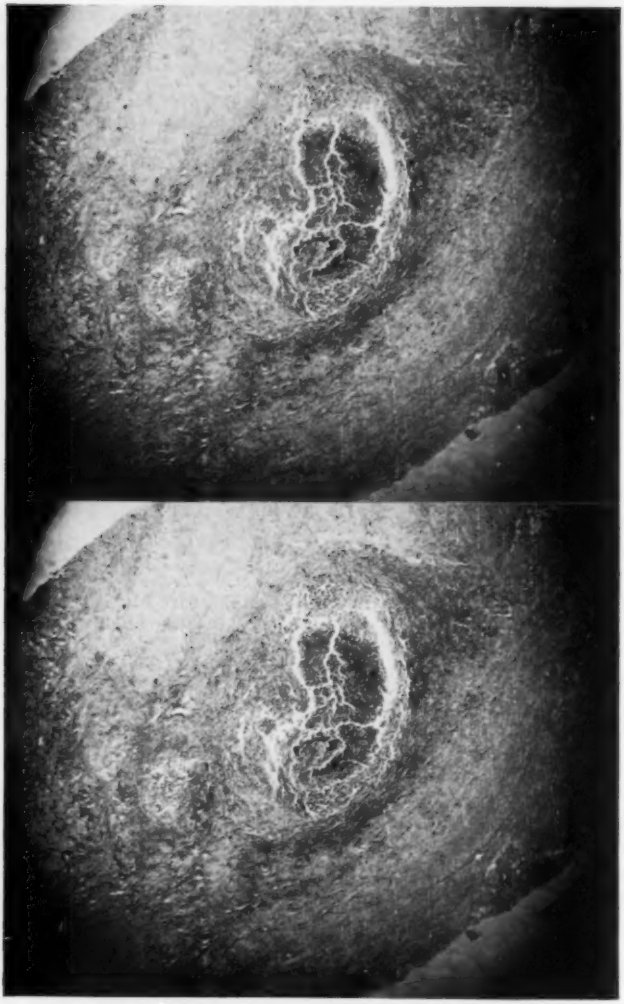

Fig. 3. Microscopic examination revealed many areas of typical Actinomy cosea with the characteristic sulfur granule arrangement.

tic histological response, although experience has also proven that different organisms may stimulate reactions that are histologically indistinguishable and, on the other hand, that a given species of micro-organisms can produce a wide variety of histological patterns. These authors for some time had made extensive bacteriological examinations of tissues removed surgically, as well as the histological examinations of the tissues removed. These studies revealed many discrepancies that occur between the histological interpretation and the results of the bacteriological studies of the same tissue.

Briefly, their method of making these bacteriological studies is to have as much of the tissue as possible removed by the surgeon under sterile technique, if possible. They preferred to have abscesses aspirated rather than to use swabs. The tissue should be ground to a paste in a sterile mortar and diluted with sterile saline or broth to make a 10 to 20 per cent emulsion. It is not satisfactory to use pieces of tissue for culture or inoculation. This emulsion is then cultured on the various media that may be required, which any modern bacteriological laboratory will be able to do.

The following is a brief summary of the conclusions of these authors:

"Tuberculosis was frequently not recognized from the biopsy alone, but bacteriological studies of the same tissue gave an accurate diagnosis. It frequently may be impossible to establish a diagnosis of blastomycosis or coccidioidomycosis by histological methods alone because of the absence of organisms, or because the organisms present may not possess adequate identifying characteristics. Brucellosis may be recognized in the tissue only by isolating the organism, as it gives no characteristic histological pattern. Actinomycosis is probably frequently confused with certain fungus infections, but careful attention to the size of the filaments would many times avoid this error; however, actinomycosis can be distinguished with certainty from nocardia only on culture. Because of the inherent limitations of histological procedures, we believe that every specimen removed for biopsy should be studied as thoroughly as possible by acceptable bacteriological methods in an attempt to establish the etiological agent. Such procedures, if performed diligently and carefully, should result in more accurate diagnoses

and thereby prevent needless operations and save many lives by indicating the proper therapy."

COMMENT.

It was very disconcerting to treat a patient for three months without making a diagnosis. Had we made bacteriological studies of the tissue taken for biopsy we might have made the diagnosis before death, and, as penicillin is effective in the treatment of actinomycosis, we might have saved his life.

CONCLUSIONS.

1. A case report of a patient who had actinomycosis of the ear with intracranial complications that caused his death in three months, has been presented.

2. The diagnosis was not made until the autopsy findings were complete.

3. Biopsies taken from the middle ear, mastoid antrum and nasopharynx revealed a granuloma.

4. Bacteriological studies made from granulomatous tissue are necessary to make a differential diagnosis between the various diseases that produce such granulomatous tissue.

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THE ADENOID AND ITS RELATION TO THE EAR.*†

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It is significant that the term adenoid merely means resembling a gland, while the term adenoids, by the simple addition of the letter "s", implies a pathologic state.

Adenoid disease has been recognized since the time of Hippocrates. Meyer of Copenhagen, when he published the classic description of adenoid vegetations in 1868, called attention to the "adenoid facies" exhibited in portraits and busts several centuries old. It is curious, therefore, that this disease and its management should be so generally neglected, both in the teaching of otolaryngology and in textbooks on the subject. The neglect, however, helps to explain why the removal of the adenoids is looked upon as one of the simplest and easiest of all surgical procedures, which it is not; in fact, the generally casual attitude toward the operation helps to explain, in its turn, why the results which follow it are so often less than satisfactory.

The adenoid was once thought to be a completely functionless structure. There is now an increasing belief that the growing child requires nasopharyngeal lymphoid tissue for present and future protection against infection. When, however, adenoid tissue has become hypertrophied, it not only cannot fulfill its normal function, but it also serves as a focus of infection.

PATHOLOGY.

The most important subject in diseases of the pharynx is the pathologic changes in the lymphoid tissue. There are two principal types of pathological changes to which the adenoids (lymphoid tissue) are subject: *a.* Hyperplasia; *b.* Hypertrophy with infection.

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In hyperplasia (see Fig. 1), which usually occurs in infants and young children, there is a marked reproduction of the normal lymphoid cells with very little, if any, of the growth of

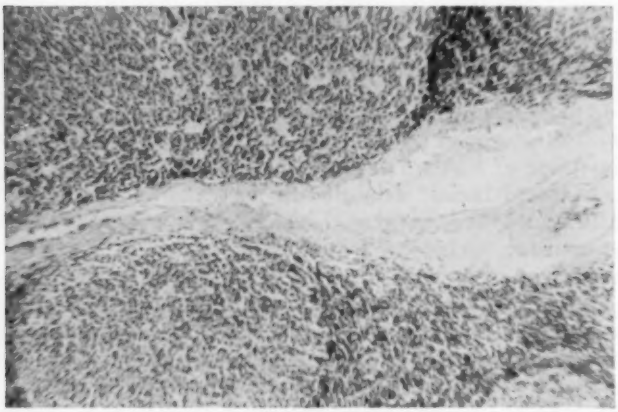

Fig. 1. Demonstrates simple hyperplasia showing prominent enlarged follicles.

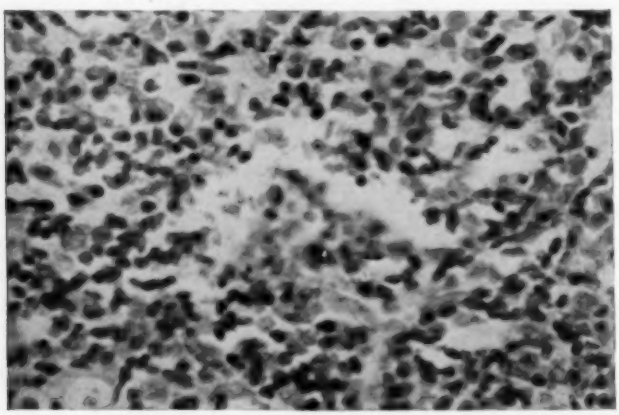

Fig. 2. Shows hypertrophy with infection. This shows chronic inflammation with many small congested blood vessels and increasing amount of fibrous tissue. Pycnosis is present (this means degeneration of the cells that stain black). Exudate and degenerated material in cells of the adenoid are also present in this illustration.

connective tissue (see Fig. 2). This tissue is subject to infection, and then the second type of disease occurs, in which a marked increase in vascularization takes place, with the other elements of inflammation, such as round cell infiltration and fibrous tissue formation. The folds within the lymphoid masses

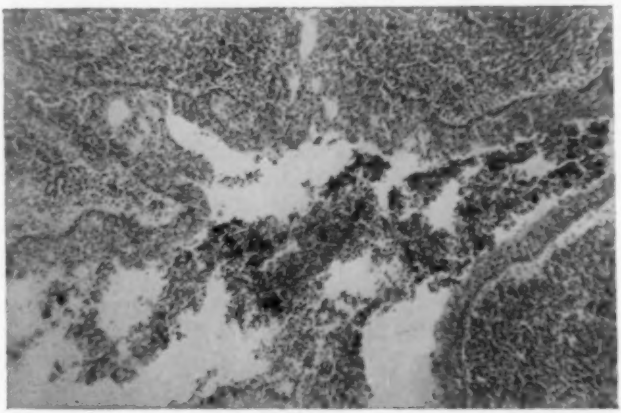

Fig. 3. Shows a crypt of the adenoid with distention of the crypt and an exudate present; upper respiratory epithelium was also present.

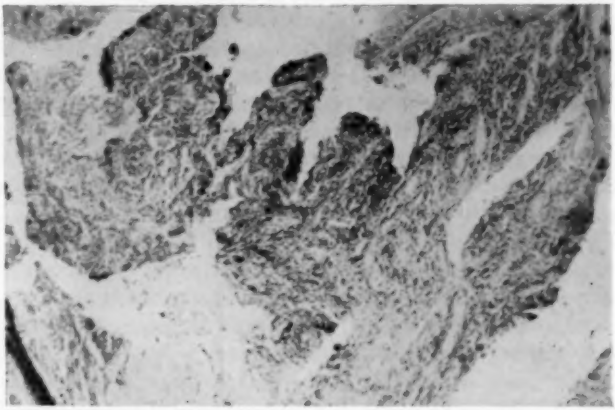

Fig. 4. Chronic inflammation, fibrosis and pycnosis are present; few lymphoid cells; fibrous tissue degenerating; cysts spaces.

in the adenoid harbor the infected material. The bacteria in the crypts find their way into the follicles. As the process of recurrent acute inflammation in the crypts and lymphoid tissue occurs, so does the retention of the infectious material within the crypts increase (see Fig. 3). This retention causes these crypts to dilate with accumulated masses of degenerated epithelium in the crypts, clumps of bacterial flora, particles of food and mucous (see Fig. 4). Slowly this hypertrophic process produces atrophy by squeezing out the lymphoid tissue. The toxins from the crypts (distended) or bacteria are absorbed into the circulation by the blood stream, or lymphatics. A marked anemia develops from diseased adenoids.

AUDITORY IMPAIRMENT.

The presence of diseased adenoids may be followed by a number of serious consequences, some infrequent but some very frequent indeed. They include snuffling, nasal obstruction, mucopurulent or purulent nasal or postnasal catarrh, absence of nasal resonance, laryngeal stridor, suppuration of the accessory nasal sinuses, cervical adenitis, gastrointestinal disturbances, defective growth and backwardness, aprosexia or inability to fix the attention, neuroses, systemic infections, and a number of other unfortunate results.

In particular, neglect of diseased adenoids may have an extremely serious effect upon the structure and functions of the ear. Deafness which follows obstruction to the Eustachian tube by adenoid tissue may occur by several mechanisms: the membranum tympani may be indrawn or retracted; postnasal catarrh may spread along the Eustachian canal, with resulting acute and chronic otorrhea. From the aural point of view, the most dangerous variety of diseased adenoid tissue is the soft, diffuse type, which usually is not excessively hypertrophied. This type of tissue more than any other type, is apt to become inflamed and suppurative, and to cause middle-ear disease and eventual auditory impairment.

These are not imaginary or even occasional possibilities. They are serious considerations which must be constantly

borne in mind. It has been said with good reason, that a definite loss of hearing can practically and without difficulty always be demonstrated in a child with hypertrophied adenoids who is not properly treated before the age of 10 years. One British observer reported diseased adenoids in 62 per cent of 375 school boys ranging in age from 6½ to 12½ years, 27 per cent of whom had some impairment of hearing. Another observer found auditory damage in 70 per cent of all children with diseased adenoids, while still another found that 60 per cent of all hearing impairment in children could be attributed to this cause.

These hearing difficulties arise for the most part during the period of active growth. Even if atrophy of the hypertrophied adenoid tissue should take place by natural processes, the regression cannot be counted on to occur before the child's hearing is in danger of being irreversibly damaged. It should also be remembered in this connection that the atrophy of the adenoid tissue which so often seems to occur at the age of puberty is usually only apparent; the adenoid tissue itself does not shrink, it merely seems smaller because, except in occasional cases, the nasopharynx has increased in size.

The otolaryngologist, confronted with the question of whether or not to operate for adenoids, often has a difficult decision to make. There are two circumstances, however, in which his course of action is clear: *1.* If enlarged adenoids are present in association with chronic suppurative otitis media, they should be removed without delay, even at the risk of an acute flare-up of the aural disease. *2.* If auditory impairment is present in association with even minimum adenoid vegetations, an operation to remove the adenoid tissue must be undertaken without delay.

EXAMINATION OF THE NASOPHARYNX.

Examination of a child suspected of harboring hypertrophied adenoids is not always easy; in fact, examination with the mirror is practically impossible in children under six years of age, and if the symptoms and clinical signs are not conclusive, examination with the finger may be necessary. If the finger is passed up behind the soft palate, a soft mass, which feels almost like a bundle of worms, may be found filling, or

almost filling, the nasopharynx. When the adenoids are diseased, the finger, when it is withdrawn, will be covered with tenacious mucus streaked with blood. If no blood is present, it can be assumed that the nasopharynx is normal. If digital examination can possibly be avoided it is well to omit it in young children, particularly nervous children, to whom it is always very disturbing.

Posterior rhinoscopy should be employed routinely in older children. If adenoids are present, the amount of enlargement can be determined by the extent to which the upper portion of the posterior edge of the septum is hidden from view in the mirror. In extreme hypertrophy the entire septum is invisible. Nasopharyngoscopy should also be performed routinely in all older children.

A child with adenoids is likely to present such findings as a high-arched palate; irregular eruption of the second teeth, which project forward and overlap; and blobs of mucus or a mucopurulent discharge dripping from the postnasal space.

TECHNIQUE OF REMOVAL OF THE ADENOIDS.

Unless the removal of the adenoids is carried out with careful attention to technical details, portions of the hypertrophied structure will inevitably be left *in situ*, particularly in the following locations:

1. The angle formed by the roof and posterior wall of the nasopharynx. One frequently finds, when examining the patient after operation, that while the main mass has been removed, a fringe of tissue has been left behind.

2. Posteriorly, behind the soft palate. When the soft palate is raised, a tag of tissue attached to the mucosa is likely to be found. Sometimes it is large enough to be seen without lifting the palate.

3. Laterally, in Rosenmüller's fossae, above and below the Eustachian cushions. When the adenoids are soft and diffuse, tissue is particularly likely to be left behind in this location.

The operation is performed under general anesthesia, which is continued in the deep plane, with abolition of the pharyn-

geal reflex, until the whole procedure has been concluded. The patient is in the recumbent position, with the head down and slightly flexed. A mouth gag is used, as in tonsillectomy.

Opinions differ as to the most useful instruments for removal of the adenoids. There is rather general agreement that a curette is preferable to the various kinds of postnasal forceps formerly employed, because it removes the growth more completely, as well as more quickly. An essential step in the curette technique is the use of a horizontal incision, made with a knife, through the mucosa immediately below the adenoid. This precaution prevents stripping of the nasopharyngeal mucosa as the curette is removed. Otherwise, surgeons tend to develop their own preferences. Some prefer the Beckmann-Thomson curette, others the LaForce adenotome, which works on the guillotine principle and which is fitted with a cage which receives the excised tissue and prevents its lodging in the larynx or being swallowed. Still others prefer the LaForce and Kelly adenotomes used in combination.

The following technique is simple and satisfactory:

1. Before any operative procedure the nasopharynx is palpated digitally, to determine accurately the size, character and position of the adenoids. In particular, it is noted whether the fossae of Rosenmüller are clear, whether the Eustachian cushions are normal or enlarged, and whether the atlas is unusually prominent.

2. The central mass is attacked first, with the LaForce adenotome. Usually this step removes only the upper two-thirds.

3. The Kelly direct vision adenotome is next inserted and the lower part of the adenoid mass is cleanly removed. Any lateral portions are also removed under direct visualization.

4. Finally, the Love retractor is inserted, and the fossae of Rosenmüller are inspected. The Yankauer speculum may also be used for this purpose. Any lymphoid tissue remaining in these areas is removed with a punch forceps. It is sometimes useful to use a Eustachian catheter at this stage of the operation.

5. The last step is a systematic inspection of the whole nasopharyngeal area, including first the roof, then Rosenmüller's fossae and the Eustachian cushions, and finally, the posterior wall. If the operation has been correctly performed and all adenoid tissue has been removed, the entire surface will be perfectly smooth.

Bleeding is sometimes so brisk during removal of the adenoids as to hamper the procedure. It seldom fails to stop, however, within a minute or two of the conclusion of the operation, and under normal circumstances, the total loss is not more than one or two ounces. If bleeding does not cease spontaneously, a thorough examination must be made at once, under good illumination, to see whether a tag of lymphoid tissue has been left behind. It is astonishing how often profuse bleeding occurs from this simple cause, and how promptly it ceases when the tag has been removed. If bleeding still continues after the removal of the tag, the quickest and most effective way to control it is to insert a gauze plug into the postnasal space. In some cases it is necessary to suture.

POSTOPERATIVE BLEEDING.

In cases of postoperative bleeding, a postnasal pack is inserted in the usual manner, making sure that you have a string from the pack coming out of the mouth and attached to the cheek. This string coming from the mouth is essential for easy removal the following day. A pack should never be left in place over 24 hours. If bleeding recurs after the pack is removed, it is much better to put in another pack than to leave the original pack in place. A pack in place over 24 hours invites secondary infection. Hemostatic drugs are also useful in controlling minor postoperative bleeding.

RECURRENCE.

When operation is performed carefully, by the technique described or some similarly precise technique, recurrence of adenoid tissue does not take place in more than 10 per cent of all cases. Some recurrences are undoubtedly instances of physiologic hypertrophy of remnants of adenoid tissue rather than true regrowth. No treatment is necessary in this type of recurrence. If, however, one is dealing with a true regrowth,

which, because of its size or location or both, is producing symptoms, the preferable method of management is secondary surgery. The symptoms that we encounter from adenoid remnants include: *1.* conductive deafness; *2.* recurrent colds; *3.* tinnitis (a subjective symptom); *4.* pressure and ear sensation (a subjective symptom); and *5.* aerotitis.

It is almost impossible to examine the adenoid region in young children with a nasal pharyngoscope or laryngeal mirror; therefore, to be positive in your diagnosis, it is wise to give the patient an anesthetic and examine the nasal pharynx thoroughly. Where tissue is found we remove enough to ventilate the Eustachian tube and the nares. Often hidden infection is found in the pharyngeal recess as small cysts. Head colds frequently start in this area. Remember to remove the tissue around the torris by nibbling with the punch forceps.

On the surface, irradiation seems a satisfactory method of managing recurrent hypertrophy of the nasopharyngeal lymphoid tissue, since lymphoid tissue ranks next to the sex cells in its sensitivity to this physical agent. On the other hand, this method is not without danger, and reported results vary widely. It seems wisest, therefore, to reserve it for small lymphoid accumulations in and around the Eustachian tubes in patients whose chief complaint is conductive deafness. Before it is resorted to, it is essential that the diagnosis of conductive deafness be established; more than one patient with nerve deafness has been submitted to unnecessary irradiation because the proper diagnostic methods were not first employed.

X-RAY TREATMENT.

The X-ray treatment is of value in real young children in whom you are sure no large mass of adenoid tissue is present. The technique for X-ray treatment of the Eustachian tubes or lymphoid tissue in the nasal pharynx is as follows:

The output of the machine (with these factors): 48 Roentgens (r) per minute. The treatment is through two fields, a right and left lateral, each 7x7 cm. in size located to crossfire the nasal pharynx. The treatment is one weekly for four weeks with both right and left fields treated at weekly intervals. The treatment to each field each week is two minutes

(*i. e.*, 96 r. air), or total air dose of 384 r. (air) to each port (field). The aim of the treatment is to give a calculated depth dose of between 300 and 400 r.'s in the midline of the pharynx.

RADIUM TREATMENT.

Radium treatment is used in older children, and the monel metal applicator is used. It is important to apply the radium applicator so that the tip of the applicator will fit directly over and in contact with the orifice of the Eustachian tube. The dose is 50 mg. of radium for 12 minutes each side (36,000 mg. per second). Seventy-five per cent of the Beta rays are absorbed in the first three millimeters of tissue. Never use the radium applicator for longer than 12 minutes, and never give more than four treatments in a period of six weeks, and no repeats. You must treat before irreversible changes occur in the middle ear. With the radium we destroy the trigger zone and lymphoid tissue that acts as a focus of infection; this contributes toward raising the patient's resistance, and the patient has fewer colds. It is important to remember the following: *1.* Use radium only in selected cases; *2.* Place radium in its proper place; *3.* Treat early before irreversible changes occur; *4.* The duration of symptoms is important in the prognosis; *5.* To get the best results, use in conductive deafness, tinnitis, pressure in the ear, aerotitis, and repeated cold cases.

Improvement in hearing follows proper attention to the nasal pharynx only in conductive deafness; however, improvement in hearing can occur in the absence of any form of treatment (spontaneous).

RESULTS.

There are few surgical procedures in which results are as good as they are after surgery of the adenoids providing *1.* that the operation is indicated and, *2.* that it is properly performed. The dull listless and even stupid-looking child becomes bright, active and healthy-appearing, with a new enjoyment of life. The hearing impairment caused by obstruction of the Eustachian tubes by adenoid tissue also promptly disappears since, when operation is performed promptly, this type of deafness is reversible.

SUMMARY.

Removal of the adenoids should be advised only when there is no doubt that the hypertrophied lymphoid tissue is producing symptoms, since it is now known that the growing child requires this type of tissue for present and future protection against infection. If, however, it can be established that impairment of health is related to the presence of adenoids, operation should be performed promptly. It should also be performed without delay if auditory impairment is associated with even minimum adenoid vegetations. A precise technique is essential. The most important cause of recurrence is inadequate surgery at the original operation. If postoperative recurrence of nasopharyngeal lymphoid tissue gives rise to symptoms, secondary surgery is usually preferable to irradition.

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Secretary of Ophthalmology Section: Richard Kratz, M.D.

Chairman of Otolaryngology Section: Harold Boyd, M.D.

Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.

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Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

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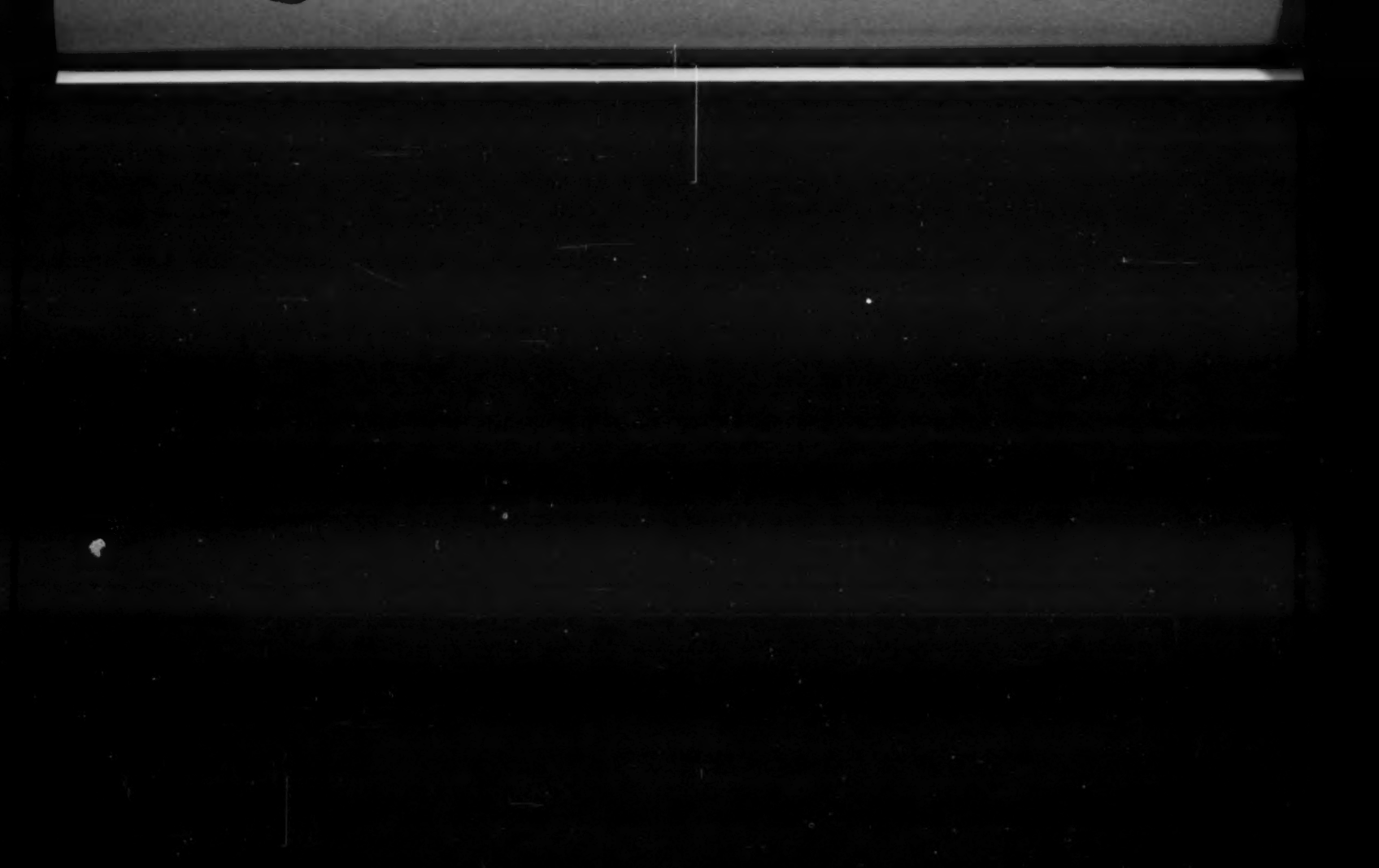

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